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GENERAL PRACTICE SERIES

THE TREATMENT OF HEAD INJURIES IN RURAL AREAS

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In the more remote areas of South Africa, where facilities are limited, the treatment of a severe head injury may present a most difficult problem. Ideally these cases should be treated in a neuro-surgical centre, but the long distances involved, and the deplorable state of many of our country roads, may necessitate treatment of the patient on the spot, despite the limited facilities, and perhaps the limited experience, of the practitioner. This state of affairs was well illustrated by Laubscher,[1] who was forced to deal with a chronic subdural haematoma in a man who had been unconscious for 18 hours in one of the more outlying areas of South West Africa. The skilful manner in which he dealt with this emergency, and the brilliant result obtained, is an indication of what can be done for the patient under these circumstances. Although most of the standard surgical text-books contain a chapter dealing with head injuries, the management of the case and the treatment recommended, is usually based on the assumption that the patient is either in, or only a short distance from, a specialized centre. Consequently, text-book descriptions are not a great deal of help to the isolated practitioner who may have to make do with the limited facilities at his disposal.

The aim of this article is to present a practical guide to the management of head injuries occurring in areas where skilled neuro-surgical help is not available. No reference will be made to special methods of investigation such as carotid angiography and air studies, since these diagnostic measures can be performed with safety only in specialized centres. Many of the life-saving operations which require to be performed in head injuries, are relatively simple, and to allow a patient to die without attempting the evacuation of a subdural or extradural haemorrhage, simple because skilled assistance is not available, is tragic indeed. We are of the opinion that the doctor who performs his own appendicectomies, is equally capable, if required, of dealing with certain head injuries which may otherwise be fatal in the absence of treatment. Let it be clearly emphasized that we are not advocating the 'burring' of skulls all over the country. Should it be at all possible, transportation of the patient to a neuro-surgical or surgical centre, experience in dealing with these cases, is mandatory.

TRANSPORT

Should it be decided to transport the patient, it is of the greatest importance to ensure that a clear unobstructed airway exists. If there is the slightest doubt on this score, tracheostomy should be performed. This comparatively minor procedure may be life-saving. An adequate description of the operation is given in most of the surgical text-books. The position in which the patient travels is likewise of great importance. The best position is probably that described by Campbell and Whitfield.[2] 'The unconscious patient should be placed on his side with the chest supported in front by a pillow. The thighs and legs are flexed, with a pillow between the thighs if necessary. The arm beneath the patient should be placed in front rather than behind, since in this position he is less likely to roll forward. The head is turned so that the mouth is slightly lower than the occiput to promote drainage of secretions. The patient is turned at regular intervals'. It is impossible to over-emphasize the importance of the measures described above, and failure to pay adequate attention to them may result in the needless death of the patient through suffocation *en route*.

OPERATIVE TREATMENT

The minimum instruments required to deal with conditions such as depressed fractures and subdural and extradural haemorrhages are shown in Fig. 1. It is urged that practitioners who may be called upon at any time to deal with

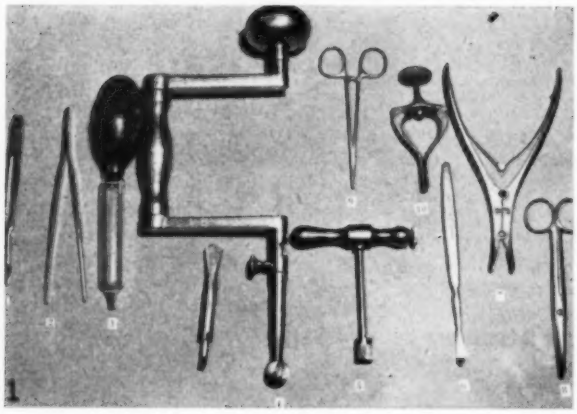

Fig. 1. 1. Scalpel. 2. Dissecting forceps. 3. Dakins' syringe. 4. Hudson's brace with perforator and burr. 5. Trephine. 6. Periosteal elevator. 7. Bone nibbler. 8. Scissors. 9. Artery forceps. 10. Self-retaining retractor.

emergencies of this nature, should take steps to ensure that these instruments are available in their local hospitals.

When a head injury is seen *within* 48 *hours* there are, in general, two indications for operating.

1. An *open scalp wound*, with or without a depressed fracture. A depressed fracture, when the skin is unbroken, is not an emergency and can be safely left and transferred to a specialist centre. These indications do not require further description.

2. *Extradural haematoma.* This condition, although occurring much less frequently than subdural haematoma, is important, since if not treated early, the patient usually succumbs, while the results of early evacuation are excellent. The existence of an extradural haemorrhage should be suspected when, after a certain degree of recovery from the original concussion, a progressive lowering in the level of consciousness occurs, i.e. a *lucid interval* is noted. As blood accumulates in the relatively confined extradural space, the intracranial pressure increases, drowsiness and restlessness become more marked, and the patient gradually lapses into coma. With the rise of intracranial pressure, localizing signs may be noted. Convulsive twitchings of the opposite side may occur and, later, a flaccid paralysis develops. If operation is not performed and the intracranial pressure is allowed to increase, bilateral motor effects may appear. The pupil may dilate on the side of the injury. It should be remembered that a fixed dilated pupil is generally a late sign and is of grave prognostic significance. When one or all of these signs are noted to have developed, an exploratory burr-hole should be undertaken immediately, for the prognosis becomes much worse if coma is allowed to supervene. To make a burr-hole on the mistaken assumption that the patient has an extradural haemorrhage will do no harm, but it is certain that a life will be lost if this is not done in the patient who has a clot. Operation is justified on the *suspicion* of extradural or subdural haemorrhage.[3]

A patient seen *more than* 48 *hours* after sustaining a head injury may be suffering from either of the two conditions mentioned above. However, a subdural haematoma is the more likely cause of cerebral compression presenting later than 48 hours after a head injury. In this condition too, a simple operation may be life-saving.

Subdural Haematoma

This condition may develop immediately after a head injury, or progress by accumulation of fluid in the subdural space during a period of days, weeks, or months. A subdural haematoma of less than 48 hours standing which produces coma or deep stupor is unlikely to be helped by operation of any type, although occasionally, through a large craniotomy, the active bleeding can be stopped, the clotted blood evacuated, and a life saved by the decompression so effected. After 48 hours, the haematoma becomes more fluid, and evacuation is possible through simple burr holes. As the days go by, the haematoma becomes more and more fluid and easier to evacuate. The ideal time for operation is between the 8th and 14th days. Thereafter a membrane forms about the fluid clot, and this sets the surgeon a further problem.

This variety of brain-surface haemorrhage is much commoner than extradural haemorrhage and the clinical picture is much less clear cut. The presence of a subdural haemorrhage should be suspected in a patient who has complained of a headache for some days after a head injury, and then becomes progressively more drowsy. A typical feature of this condition is fluctuation in the level of consciousness. The latent interval is generally much longer than in extradural haemorrhage, and it may take many days for signs of compression to occur. Localizing signs are more likely to be absent than present. It is important to realise that subdural haematoma may follow a relatively mild head injury. Again, exploration should be performed if there is the slightest possibility that the condition has occurred.

OPERATIVE TREATMENT

Anaesthesia

Local infiltration of the proposed incision in usually sufficient. 1% Xylocaine with adrenaline (1:200,000) is advised, but 1% novocaine with adrenalin can be used. A general anaesthetic may be necessary with a violent patient, and this should be combined with local infiltration of novocaine and adrenalin, the latter aiding haemostasis.

1. *Open Wounds*

Clean-cut incised wounds should be sutured. A ragged wound should be cleaned, the edges excised, and apposition secured.

2. *Compound Depressed Fracture*

After preliminary cleaning, the skin edges should be excised, and the wound extended so that the whole extent of the fracture can be seen. A burr-hole is made next to the fracture in healthy bone and, from this point, bone is nibbled away with a bone nibbler so as to encircle the depressed area. Schrire[4] has suggested the use of a dental forceps as a substitute for a bone nibbler should this not be available. The depressed fragments are now free, and can be elevated and removed If the dura is torn, it should be sutured. The scalp should be closed in 2 layers with silk or catgut, and dermalon used for the skin.

3. *Extradural Haemorrhage*

The side chosen for exploration is that of the dilated and perhaps inactive pupil, should this be present. If a hemiplegia has occurred, the opposite side is chosen. If X-rays are available, the existence of a fracture line will probably localize the haematoma to that side. Where there is no difference between the two sides, it may still be possible to make the diagnosis if the sequence of events are known.[5]

After the skin has been shaved and cleaned, a 2-3 inch vertical incision is made one finger's breadth in front of the ear. The lower end extends to the malar bone (Fig. 2). Active bleeding points are coagulated or tied (the superficial temporal artery is usually encountered). The incision is deepened through the temporalis fascia and temporalis muscle to the bone. Bleeding points are again dealt with. The temporalis muscle is scraped off the bone (squamous temporal) with a periosteal elevator or blunt dissector, and a self-retaining retractor is inserted. A burr-hole is now made, first with a perforator or a small burr, the hole then being enlarged with a larger burr. A trephine, whilst more dangerous and cumbersome, can be used instead.

If an extradural haematoma is found, no attempt should be made to remove it until the burr-hole is enlarged by nibbling bone away from the burr-hole outwards by means of a bone nibbler or perhaps a dental forceps. When the bony defect is about 1½ inches in diameter the clot can be evacuated, preferably by suction, or by mopping and washing away with warm saline. If an active bleeding point is encountered, e.g. the middle meningeal artery, it should be coagulated, tied off, or sutured. If the clot is found to extend backwards, and bright-red blood wells up in the wound from this direction, it is probable that the posterior branch of the middle meningeal artery is bleeding. Under these circumstances it is best to make another vertical incision above and behind the ear and to open the skull about 1½ inches above and behind the external auditory meatus. It is uneccessary to search for a torn vessel if, after removal of the clot, there is no further haemorrhage.

The rough bony edges, which tend to bleed, should be filled with bone wax if available. If a trephine has been used, the block of bone removed, should not be replaced. The temporalis muscle is now approximated with silk or catgut sutures, it is advisable to drain the wound by means of a glove drain inserted through a separate incision. The skin should preferably be closed in 2 layers.

4. *Subdural Haematoma*

Again the side chosen is that of the affected pupil and contralateral to the hemiparesis. Two incisions are made. The first (anterior) incision is about 1½ inches in length, just within the hairline and 2 fingers' breadth lateral to the mid-line (Fig. 2). The pericranium is divided with the skin, and scraped off the bone (frontal) with a periosteal elevator. A hole is made with the perforator and burr, or by means of a trephine, as described above. The dura exposed at the bottom of the burr-hole is preferably touched with the coagulating diathermy, if this is available. If a subdural haematoma is present, the dura will be bulging and dark in colour because of the blood beneath it. The dura is now elevated with the point of a curved needle on a needle holder and, with the dura thus lifted away from the brain surface, it is incised in a cruciate manner. The black subdural haematoma will now spurt out, and the opening in the dura is enlarged to the extent of the burr-hole.

The second incision is now made on the same side, above and behind the ear 3 fingers' breadth lateral to the mid-line and at right-angles to it (Fig. 2). A burr-hole is again made, and the dura opened in the manner described. Fluid blood, if present, will escape. The space now apparent should be thoroughly washed out with warm saline (98·4°F) until it

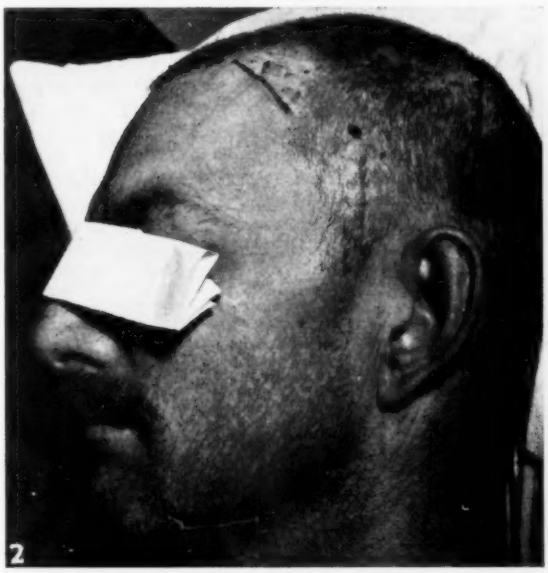

Fig. 2. Anterior and posterior incisions for subdural haematoma. The middle incision is that used for extradural haemorrhage.

returns clear through both burr-holes. A catheter (size 4 or 5) can be introduced through each burr-hole in turn in all directions, and warm saline again injected through it until the return is clear. At this stage the head of the table is lowered so as to assist evacuation of the haematoma and also to aid the compressed brain to assume its normal position near the dura. A rubber catheter can be left through each burr-hole to allow any further fluid to escape during the next 24 hours. It will be noted that no attempt is made to locate the bleeding vessel.

The wounds are sutured, preferably in 2 layers, with silk or catgut, and dermalon. The catheters are secured to prevent them from slipping into the wounds.

A frontal burr-hole is now made on the contralateral side, whether a subdural haematoma has been found on the first side or not. This should always be done, as the wrong side for the first burr-hole may have been chosen, the clinical findings being ipsilateral, and because a subdural haematoma is itself not infrequently bilateral. If a haematoma is not found in this situation the incision may be closed, but should a subdural haematoma be present a posterior incision must be made and the operation proceeds as described above.

Post-operatively the patient should be treated in a slight Trendelenberg position. A clear airway is vital for the recovery of the patient. The unconscious patient easily becomes

'moist' and frequent cleansing of the air passages by suction should be done. Even in the hands of nursing staff experienced in dealing with these cases, the patient may become progressively more obstructed and, if there is the slightest doubt concerning the adequacy of the airway, tracheostomy should be performed.

SUMMARY

1. In the more remote areas of the country, practioners may be called upon to perform emergency operations on certain types of head injury.

2. Operative methods are described for dealing with depressed fractures and extradural and subdural haemorrhage, and the relative simplicity of these operations is stressed.

3. Factors considered important in the safe transport of the unconscious patient are discussed.

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EDITORIAL

LUNG CANCER AND SMOKE

The statistical link between lung cancer and cigarette smoke is no longer disputed.[1] Present trends of research seem to be aimed at establishing the relative place of inhaled smoke and its carcinogenic components in the aetiology of the disease, rather than questioning it. Whilst tobacco is probably the most important source of carcinogen-containing smoke in South Africa, the polluted skies over the industrial cities of the Northern Hemisphere are scarcely less of a threat. On both sources, viz. tobacco and a polluted atmosphere, a good deal of research has been reported in recent years.

Evidence of illness and death from 'smog' is undisputed but the effects of continued exposure to lesser degrees of pollution are less certain. The fact that an environmental factor exists in lung cancer has been known for a decade or more, and this is clearly bound up with the amount of smoke in the atmosphere. Kennaway (amongst others) showed differences existing between death rate in town and country,[2] and Stocks wrote in 1947: 'Either smokiness of the atmosphere is an important factor in itself in producing carcinoma of the lung, or sunshine is an important factor in preventing its incidence.'[3] Meanwhile other research workers demonstrated the presence of a carcinogenic hydrocarbon in cigarette smoke[4] and also in the atmosphere of most places,[5] though no quantitative survey was carried out and the concentration relative to the degree of pollution present in the air was not worked out. In order to do this, a research team under Stocks and Campbell[6] is at present comparing two areas in the north of England—one in the heart of Liverpool, an almost permanently smoke-bound city, the other a semi-urban community in Cheshire—with a wholly rural part of North Wales. Each notified case of cancer is carefully investigated and the environmental history as well as the previous smoking history of the patient is noted. The histories of over 10,000 males between the ages of 45 and 75 with lung cancer were collected in this way over a period of 2 years. (The actual survey is to run over a 5-year period, but the conclusions were already so startling and clear-cut after 2 years that the team decided to publish their interim report.[6]) At the same time chemical analysis of the contents of the atmosphere was carried out at 10 different stations in the survey zones over a period of 6 months. Particular attention was paid to the presence and

VAN DIE REDAKSIE

LONGKANKER EN ROOK

Die statistiese skakel tussen longkanker en sigaretrook word nie langer betwis nie.[1] Dit skyn of huidige neigings by navorsing daarna streef om die relatiewe aandeel van ingeasemde rook en die karsinogeniese bestanddele daarvan by die etiologie van die siekte vas te stel eerder as om dit in twyfel te trek. Terwyl tabak waarskynlik die belangrikste bron van karsinogeen-bevattende rook in Suid-Afrika is, is die besoeldelde lug van die industriële stede van die Noordelike Halfrond amper net so 'n groot bedreiging. Gedurende die afgelope jare is heelwat navorsing in verband met beide bronne, nl. tabak en besoedelde lug, aangemeld.

Bewyse van siekte en sterfgevalle weens ‚smog' kan nie bestry word nie, maar die uitwerking van voortdurende blootstelling aan geringer grade van besoedeling is minder seker. Die feit dat daar 'n omgewingsfaktor by longkanker bestaan, is reeds vir 'n dekade of langer bekend en dit is duidelik dat dit met die hoeveelheid rook in die atmosfeer saamgaan. Kennaway (onder andere) het getoon dat daar verskille tussen die sterftesyfer in stad en platteland bestaan,[2] en in 1947 het Stocks geskryf: ‚Either smokiness of the atmosphere is an important factor in itself in producing carcinoma of the lung, or sunshine is an important factor in preventing its incidence.'[3] Ondertussen het ander navorsers die teenwoordigheid van 'n karsinogeniese koolwaterstof by sigaretrook, asook in die lug van die grootste aantal plekke, gedemonstreer[5] alhoewel geen kwantitatiewe ondersoek gedoen is, en die konsentrasie relatief tot die mate van besoedeling in die lug aanwesig, nie uitgewerk is nie. So dat dit gedoen kan word, is 'n navorsingspan onder die leiding van Stocks en Campbell[6] tans besig om twee streke in die noorde van Engeland—een in die hartjie van Liverpool, 'n stad wat feitlik altyd in rook omhul is, en die ander een 'n half-stedelike gemeenskap in Cheshire—met 'n heeltemal plattelandse gebied van Noord-Wallis te vergelyk. Elke aangemelde geval van kanker word sorgvuldig ondersoek en die geskiedenis van die pasiënt se omgewing, asook van sy vorige rookgewoontes, aangeteken. Die geskiedenisse van meer as 10,000 mans met longkanker tussen die ouderdom van 45 en 75 is op hierdie wyse oor 'n tydperk van 2 jaar versamel. (Die werklike ondersoek sal oor 'n tydperk van 5 jaar strek, maar na 2 jaar was die gevolgtrekkings alreeds so verrassend en duidelik-omskrewe, dat die groep navorsers besluit het om hulle interimverslag te publiseer.[6]) Terselfdertyd is die inhoud van die lug op 10 verskillende poste in die ondersoeksones oor 'n tydperk van 6 maande ontleed. Aandag is veral geskenk aan die aanwesigheid en konsentrasie van 3 : 4-benspireen in elke gebied. Die opsomming van Stocks en Campbell se interimverslag word in die 2 tabelle

concentration of 3 : 4-benzpyrene in each area. The gist of Stocks and Campbell's interim report is contained in the 2 tables printed below. In order to simplify the findings and

LUNG CANCER DEATH RATES* OF MALES 45-74 YEARS

	Liverpool (*Urban*)	*Wales* (*Rural*)
Non-smokers	514	70
Pipe-smokers	479	179
Cigarette-smokers:		
Light (up to 14 a day)	1,082	293
Moderate (15-35 a day)	997	657
Heavy (over 35 a day)	1,336	1,249

* Mean annual per 100,000 from mid-1952 to mid-1954.

emphasize the difference between town and country, the semi-urban community in Cheshire is omitted; in all readings it falls between the 2 extremes. Several observations can be made from these figures.

1. In a rural community (where atmospheric pollution is minimal) the incidence of lung cancer increases progressively with the number of cigarettes smoked. The death rate from lung cancer of heavy cigarette smokers (over 35 a day) was 20 times that of non-smokers, and 8 times greater than that of pipe-smokers.

2. The urban lung-cancer rate was much higher. Among non-smokers it was 8 times that of non-smokers in the country, but as cigarette-smoking became heavier, the difference tended to disappear.

3. There is an 'urban factor' present which helps to push up the incidence of lung cancer. What is this factor?

AVERAGE CONCENTRATIONS OF SMOKE, BENZPYRENE AND SULPHUR DIOXIDE IN ATMOSPHERE *

	Liverpool (*Urban*)	*Wales* (*Rural*)
Smoke[1]	36·3	4·9
3 : 4-Benzpyrene[2]	7·7	0·7
Sulphur Dioxide[3]	2·8	0·3

* During 6 months from 13 October 1954. 1. Milligrams per 100 c. metres. 2. Micrograms per 100 c. metres. 3. Milligrams per day collected by 100 sq. cm. of lead peroxide.

Stocks and Campbell draw attention to the close correlation between the concentrations of 3 : 4-benzpyrene in the air (about 10 times as high in Liverpool as in Wales) and the relative incidence of lung cancer in non-smokers (about 7 times as high in Liverpool as in Wales).† Apparently the concentration increases with urbanization, depending on the proximity of industry, houses and motor vehicles. Although they forbear to incriminate 3 : 4-benzpyrene as the villain of the piece, it is clear that Stocks and Campbell will show little surprise if a closer relationship is proved between this carcinogen and lung cancer.

Meanwhile Wynder of the Sloan-Kettering Institute, New York, has been investigating the origin of the carcinogens (and notably of 3 : 4-benzpyrene) in raw tobacco. He has found that the waxy coating of the leaf is the major source, and if it is removed with solvents, the tobacco is left relatively

† In the Liverpool atmosphere as compared with that of rural Wales there was also an excess of 1:12-benzperylene, pyrene, fluoranthene and anthracene.

hieronder aangegee. Om hierdie bevindinge te vereenvoudig en die verskil tussen stad en platteland te beklemtoon, is die half-stedelike gemeenskap van Cheshire weggelaat; by alle

LONGKANKER-STERFTESYFERS* VAN MANS 45-74

	Liverpool (*Stedelik*)	*Wallis* (*Plattelands*)
Nie-Rokers	514	70
Pyprokers	479	179
Sigaretrokers:		
Lig (tot 14 per dag)	1,082	293
Matig (15-35 per dag)	997	657
Straf (meer as 35 per dag)	1,336	1,249

* Gemiddelde jaarlikse per 100,000 van middel-1952 tot middel-1954.

lesings val dit tussen die 2 uiterstes. Verskeie waarnemings kan van hierdie syfers gemaak word.

1. By 'n plattelandse gemeenskap waar die atmosferiese besoedeling gering is, neem die voorkoms van longkanker progressief toe met die aantal sigarette wat gerook word. Die sterftesyfer as gevolg van longkanker by strawwe sigaretrokers (meer as 35 per dag) was 20-maal dié van nie-rokers, en 8-maal groter as dié van pyprokers.

2. Die stedelike longkanker-sterftesyfer was baie hoër. By nie-rokers was dit 8-maal dié van nie-rokers op die platteland, maar soos die rook van sigarette toeneem, was die verskil geneig om te verdwyn.

3. Daar is 'n ‚stedelike faktor' aanwesig wat help om die voorkoms van longkanker te laat toeneem. Wat is hierdie faktor?

GEMIDDELDE KONSENTRASIES VAN ROOK, BENSPIREEN EN SWAWELDIOKSIED IN DIE LUG *

	Liverpool (*Stedelik*)	*Wallis* (*Platteland*)
Rook[1]	36·3	4·9
3 : 4-Benspireen[2]	7·7	0·7
Swaweldioksied[3]	2·8	0·3

* Gedurende 6 maande vanaf 13 Oktober 1954. 1. Milligramme per 100 k. meters. 2. Mikrogramme per 100 k. meters. 3. Milligramme per dag deur 100 vk. sm. loodperoksied versamel.

Stocks en Campbell vestig die aandag op die noue verwantskap tussen die konsentrasies van 3 : 4-benspireen in die lug (ongeveer 10-maal so hoog in Liverpool soos in Wallis) en die relatiewe voorkoms van longkanker by nie-rokers (ongeveer 7-maal so hoog in Liverpool soos in Wallis).† Waarskynlik neem die konsentrasie met verstedeliking toe, wat afhang van die nabyheid van industrieë, huise en motorvoertuie. Alhoewel hulle hulself daarvan onthou om 3 : 4-benspireen as die skuldige te bestempel, is dit duidelik dat Stocks en Campbell nie juis verbaas sal wees as daar 'n nouer verwantskap tussen hierdie karsinogeen en longkanker bewys word nie.

Ondertussen was Wynder van die Sloan-Kettering-Instituut, New York, besig om die oorsprong van die karsinogene (en veral van 3 : 4-benspireen) by onbewerkte tabak te ondersoek. Hy het gevind dat die wasagtige omhulsel van die blaar die hoofoorsprong is, en as dit met oplosmiddels verwyder word, die tabak daarna betreklik karsinogeen-vry is. Nog een van sy ontdekkings mag die eienaardige feit, dat pyprokers minder

† In die Liverpoolse lug, in vergelyking met dié van plattelandse Wallis, was daar ook 'n oormaat van 1 :12-bensperileen, pireen, fluroateen en antraseen.

free of carcinogen. Another of his discoveries may explain the curious fact that pipe-smokers are less liable to lung cancer than cigarette-smokers. Cigarettes burn at 880°, and the carcinogenic tar liberated at that temperature is far more liable to produce new growths in laboratory animals than the tar produced by pipes, which burn at a lower temperature (720°). Tobacco burnt at 850° or less produced no carcinogen at all. Other points made by Wynder in his report to the American Institute for Cancer Research were:[7] tobacco types (e.g. Turkish, Virginia) may vary in their nicotine content, but the amount of carcinogen is constantly related to the weight of the cigarette; nicotine has no carcinogenic properties; and, finally, the filters that he tested were totally ineffective in removing carcinogen from the smoke inhaled.

1. Doll, R. and Hill, A. B. (1950): Brit. Med. J., **2**, 739.
2. Kennaway, E. L. and Kennaway, N. M. (1947): Brit. J. Cancer, **1**, 260.
3. Stocks, P. (1947): *Regional and Local Differences in Cancer Death Rates*. General Register Office. Studies on Medical and Population Subjects No. 1. London: H. M. Stationery Office.
4. Cooper, R. L. and Lindsey, A. J. (1955): Brit. J. Cancer, **9**, 304.
5. Waller, R. E. (1952): *Ibid.*, **6**, 8.
6. Stocks, P. and Campbell, J. M. (1955): Brit. Med. J., **2**, 923.
7. Reported in *The Times* (London), 13 April 1957.

THE DURBAN CONGRESS

ANNOUNCEMENT BY CHAIRMAN OF ORGANIZING COMMITTEE (DR. A. BROOMBERG)

The Second Circular containing full details of the arrangements which are being made for the forthcoming Scientific Meeting in Durban, will be in the post in a few days. It represents almost the culmination of the task on which my Committee started its work 15 months ago and little remains now but to perfect every last detail to ensure that it will be in actual fact the great and memorable occasion which we have planned to make it.

In the Congress News Letter which is published in this issue of the *Journal* (p. 518) will be found the names of 18 world-famous figures who will participate in the scientific proceedings.

Other men of international renown are also expected.

The Plenary Sessions have been planned on a most ambitious scale and their subject matter should be not only of the greatest interest to the whole profession, but will also without doubt have a tremendous stimulating effect on the standard of medicine in South Africa.

On the social side every effort is being made to provide entertainment and relaxation with the aid of all the unique facilities which Durban can offer.

We have told Durban to expect at least a thousand visitors, and Durban is prepared not only to receive them, but also to welcome them and entertain them and to make their stay happy, memorable and unforgettable.

INTENTION CARDS WILL BE ENCLOSED WITH THE CIRCULAR. MAY I EARNESTLY APPEAL TO MEMBERS OF THE ASSOCIATION,

geneig is om longkanker op te doen as sigaretrokers, verklaar. Sigarette brand teen 880° en die karsinogeniese teer wat by hierdie hittegraad vrygestel word, is baie meer geneig om nuwe gewasse by toetsdiere te veroorsaak as die teer wat deur pype vrygestel word en wat teen 'n laer hittegraad (720°) brand. Tabak wat teen 850° of minder gebrand is, het glad geen karsinogeen opgelewer nie. Ander punte in Wynder se verslag aan die American Institute for Cancer Research was:[7] die nikotieninhoud van tabaksoorte (bv. Turkse, Virginiese) mag varieer, maar die hoeveelheid karsinogeen is altyd verwant aan die gewig van die sigaret; nikotien het geen karsinogeniese hoedanighede nie; en, ten slotte, die filtreerders wat hy getoets het, was heeltemal ondoeltreffend om karsinogeen uit die ingeasemde rook te verwyder.

1. Doll, R. en Hill, A. B. (1950): Brit. Med. J., **2**, 739.
2. Kennaway, E. L. en Kennaway, N. M. (1947): Brit. J. Cancer, **1**, 260.
3. Stocks, P. (1947): *Regional and Local Differences in Cancer Death Rates*. General Register Office. Studies on Medical and Population Subjects No. 1. London: H.M. Stationery Office.
4. Cooper, R. L. en Lindsey, A. J. (1955): Brit. J. Cancer, **9**, 304.
5. Waller, R. E. (1952): *Ibid.*, **6**, 8.
6. Stocks, P. en Campbell, J. M. (1955): Brit. Med. J., **2**, 923.
7. Soos berig in *The Times* (Londen), 13 April 1957.

DIE DURBAN-KONGRES

AANKONDIGING DEUR DIE VOORSITTER VAN DIE ORGANISERENDE KOMITEE (DR. A. BROOMBERG)

Die Tweede Omsendbrief, wat volle besonderhede bevat aangaande die reëlings wat vir die komende Wetenskaplike Vergadering in Durban getref word, sal binne 'n paar dae afgestuur word. Dit verteenwoordig byna die hoogtepunt van die taak waarmee my Komitee 15 maande gelede begin het, en daar bly weinig oor om te doen behalwe om die laaste kleinighede tot volmaaktheid te bring en sodoende te verseker dat dit werklik die grootse en gedenkwaardige geleentheid sal wees wat ons beplan het.

In die Kongres-nuusbrief, wat in hierdie uitgawe van die *Tydskrif* (bl. 518) geplaas word, sal die name van 18 wêreldberoemde persone, wat aan die wetenskaplike verrigtinge sal deelneem, gevind word. Ander persone van internasionale vermaardheid word ook verwag.

Die Voltallige Sittings is op 'n baie ambisieuse skaal beplan en die onderwerpe daarvan sal nie alleen van die grootste belang vir die hele beroep wees nie, maar sal ook ongetwyfeld 'n geweldige stimulerende uitwerking op die standaard van interne geneeskunde in Suid-Afrika hê.

Met behulp van al die unieke fasiliteite wat Durban bied, word daar op sosiale gebied alle pogings aangewend om vermaaklikheid en ontspanning te voorsien.

Ons het Durban meegedeel om ten minste eenduisend besoekers te verwag en Durban is nie alleen bereid om hulle te ontvang nie, maar ook om hulle te verwelkom en te onthaal en om hulle verblyf gelukkig, gedenkwaardig en onvergeetlik te maak.

VOORNEME-KAARTJIES SAL BY DIE OMSENDBRIEF INGESLUIT WORD. MAG EK 'N ERNSTIGE BEROEP DOEN OP LEDE VAN

WHO INTEND TO BE PRESENT, TO RETURN THESE CARDS AS SOON AS POSSIBLE. ONLY FOUR MONTHS NOW REMAIN AND THERE IS A GREAT DEAL OF ORGANIZATION STILL TO BE DONE TO ENSURE THAT EVERY MEMBER WHO ATTENDS WILL BE ACCOMMODATED AND PROVIDED FOR IN EVERY POSSIBLE RESPECT.

DIE VERENIGING WAT VAN VOORNEME IS OM DIE KONGRES BY TE WOON, OM HIERDIE KAARTJIES SO GOU MOONTLIK TERUG TE STUUR. DAAR IS SLEGS 4 MAANDE OOR EN DAAR MOET HEELWAT REËLINGS GETREF WORD OM TE VERSEKER DAT ELKE LID WAT AANWESIG SAL WEES, GEHUISVES, EN IN ELKE MOONTLIKE OPSIG VERSORG WORD.

PULMONARY ARTERIO-VENOUS FISTULAE

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There are two fundamental differences between an arterio-venous fistula situated in the systemic circulation and one occurring in the pulmonary circuit: (*a*) Blood passing through the pulmonary arterio-venous fistula reaches the left heart and not the right heart, and (*b*) blood short-circuits the pulmonary capillary bed and therefore remains unoxygenated. Thus, all those symptoms of the systemic arterio-venous fistula which are due to the return of the large amount of blood to the right auricle, which sets up certain reflex mechanisms, and the symptoms caused by the leak in the arterial system, are absent in pulmonary arterio-venous fistulae. Amongst these are the increase in heart rate, the fall in diastolic blood-pressure, the enlargement of the right heart, and cardiac failure. Instead, pulmonary arterio-venous aneurysms present a picture similar to cyanotic heart-disease, since the blood reaching the left heart is a mixture of both oxygenated and unoxygenated blood. It follows that the conspicuous outward signs of arterio-venous fistulae of the lung are (*a*) central cyanosis, (*b*) clubbing and (*c*) polycythaemia.

Arterio-venous fistula of the lung is relatively rare. The first clinical diagnosis was made by Smith and Horton in 1939.[20] No account of the lesion had been found in the world reports of large series of routine autopsies until 1939.

The first unquestionable case appears to have been that of Rodes in 1938,[27] who described a 'cavernous haemangioma of the lung with secondary polycythaemia' in a man who had dyspnoea, clubbing and cyanosis, haemangiomata of the lips and who died from repeated haemoptysis at the age of 25.

Arterio-venous fistulae of the lung must not be confused with a systemic arterio-venous fistulae situated in the thorax. The symptomatology of the latter is obviously that of a systemic arterio-venus fistula situated anywhere else, particularly in the periphery.

Theoretically, the following arterio-venous communications are possible in the thorax:

1. Aorta to pulmonary artery (e.g. patent ductus arteriosus).
2. Aorta or any of its branches to vena cava or a systemic vein (e.g. subclavian vein); i.e. systemic arterio-venous fistula affecting the chest wall or the bronchial vessels.
3. Aorta or systemic artery (e.g. subclavian artery) to pulmonary vein.[47]
4. Pulmonary artery to systemic vein.
5. Pulmonary artery to pulmonary vein (true pulmonary arterio-venous fistula).

It is only the last type of communication that we are dealing with in this article.

Although most of these arterio-venous fistulae occur in the lung parenchyma itself, the communication not seldom occurs in the mediastinum or in the hilum of the lung and does not involve the lung tissue itself.

Some authors before Rodes[37, 54, 55, 56] described pulmonary varix and local venous dilations which may well have been arterio-venous fistulae and, although they recognized their congenital character,[56] they did not appear to be aware of the possibility of such a lesion and the accompanying physiological derangement. They therefore did not recognize the clinical entity as such. Both Churton (1897)[45] and Hedinger (1907)[33] have been credited with describing pulmonary arterio-venous fistulae because they recognized the association of the pulmonary lesion with polycythaemia, and Reading,[25] in 1932, was apparently the first to emphasize cyanosis. On scrutinizing their papers we cannot subscribe to this view. Hedinger clearly described a saccular varix (*typische sackförmige Varix*) of the right vena pulmonalis in a patient who might well have suffered from pulmonary tuberculosis. His case showed no cyanosis, no clubbing, and probably no polycythaemia, but he suffered repeated haemoptyses. Churton's case was one of multiple aneurysms of the pulmonary artery leading to haemoptysis and dropsy, which may well have been mycotic in origin, for the tricuspid and endocardium exhibited several wart-like growths.

Some authors[48, 49] make a distinction between the term arterio-venous 'aneurysm' and arterio-venous 'fistula', the latter being a direct communication between artery and vein, whereas in the former the connection is *via* false aneurysm through rupture of either an artery or a vein. As in the systemic counterpart numerous small telangiectases are often responsible for short-circuiting the blood,[50-52] besides a more definite larger channel.[53] Excellent reviews of this

subject have been written by Yates *et al.*[9] and Weiss and Gasul.[50]

GENESIS OF THE LESION

Arterio-venous fistula of the lung can be explained on an embryological basis. It is a developmental malformation and not a tumour. In the earliest intra-embryonic life, the blood vessels form a diffuse capillary plexus. In differentiating into arteries and veins, larger channels are formed by enlargement of individual capillaries, fusion and confluence of adjacent ones, whereas others undergo atrophy and are obliterated. The cause for this differentiation and elaboration is not well understood but genetic factors and local haemodynamic forces are concerned in the establishment of the final pattern. If during the course of this development some connections, which normally obliterate, remain patent, and small capillary pathways develop into larger vascular structures, abnormal arterio-venous connections may become established.

A definite familial incidence has been claimed for over half the cases.[1, 9, 41] Pulmonary arterio-venous fistulae were seen in siblings,[13, 40, 29, 60] and in more than one generation.[43, 1, 10]

The lesion has been considered as part of hereditary haemorrhagic telangiectasia,[9, 23, 28, 51] although Goetz[43] has pointed out that telangiectases are also seen in systemic arterio-venous fistulae. This need not come as a surprise, for the genesis of both systemic and pulmonary fistulae are, in the last analysis, similar.

CLINICAL FEATURES

The symptoms and signs will depend upon the size of the shunt and the amount of unoxygenated blood by-passing the capillary bed of the lung. In the well-developed case dyspnoea, haemoptysis and dizziness are the commonest symptoms. Cyanosis, clubbing and polycythaemia are the typical signs. However, with increasing knowledge of the condition, more asymptomatic cases are being described.

Cyanosis is usually present from birth, but may be delayed. A few patients have been described in whom cyanosis was absent,[3, 9, 23] as was the case in our first patient, who has been followed up for 23 years. Once manifest, cyanosis usually deepens and, although it may be as severe as in any case of cyanotic heart disease, it is often out of proportion to the disability and particularly the degree of dyspnoea. Cyanosis deepens with exercise. Three factors contribute to its depth, viz. (*a*) the shunt, (*b*) the polycythaemia, and (*c*) the increase in blood volume. However, cyanosis may occur in the absence of polycythaemia. If, owing to previous haemorrhage, there should be an anaemia, the cyanosis may be masked, only becoming apparent when the blood picture has been corrected.

Dyspnoea. Progressive dyspnoea on exertion is the most commonly observed symptom of anoxaemia. Stimulation of the respiratory centre and of the sino-aortic chemo-receptors by the high carbon-dioxide content of the arterial blood has been thought to be responsible for these symptoms.[15] It is not caused by pulmonary congestion or congestive cardiac failure, for neither is a feature of the condition.

Clubbing of the Digits. Clubbing seems to go parallel with the degree of cyanosis. As soon as cyanosis is manifest, clubbing appears. Thus our patient, as well as those reported in the literature who had no cyanosis, also had no clubbing.

Dizziness. Transient attacks of vertigo or fainting, with or without convulsions, particularly after exertion, have been observed. Although attributed by Lindgren[39] to cerebral air embolism, it is more likely that they are caused by cerebral anoxaemia, as in cyanotic heart disease. Paroxysmal nocturnal dyspnoea, substernal oppression and paraesthesiae have been attributed to the extremely low oxygen saturation of the arterial blood.[11]

Haemorrhage. Haemoptysis may be the first symptom bringing the patient to the doctor. It caused the death of Rodes' patient.[27] Bowers' case[6] died from a rupture of the haemangioma into the pleural cavity. Apart from this, haemorrhage may arise in the telangiectasis of the nasal septum, the tongue, the eyelids, and the naso-pharyngeal mucosa. Haematemesis has also been reported.[31]

Additional Symptoms. Beierwaltes and Byron[11] were the first to take note of certain peculiar and unexplained aspects of the symptomatology related to the left side of the body. They refer to the case of Adams *et al.*,[30] who could not lie on the left side because it caused him to cough, and they mention one of their own cases who could never lie on her left side because she would immediately develop intense paraesthesiae. In both cases the symptoms were relieved by surgery. A patient of Sisson *et al.*[21] developed numbness in the left arm and shoulder and weakness of the left hand shortly before admission to hospital. A case reported by Derra[31] had attacks of temporary left-sided stiffness and paraesthesiae of the left arm, and one of Lindgren's patients[39] had a temporary left-sided hemiparesis.

Physical Examination

The main features of cyanosis and clubbing have already been mentioned. Both may be absent in the mild case, may only develop later in life, or may be so gross as to suggest the severest of congenital heart diseases.

The heart itself does not, however, reveal any abnormality, there being no enlargement, no rise in blood pressure, and no increase in heart rate. There are no signs of right heart failure, as shown by the normal venous pressure. As the shunt is parallel to the pulmonary capillary system, with its low resistance, it is not necessarily associated with an increased return of blood into the left auricle. Both the lower pressure-gradient and the lower peripheral resistance in the pulmonary circulation militate against a great flow through the fistula. The cardiac output, therefore, is not increased as long as the blood volume is not increased.

The blood pressure does not rise despite the increased circulating blood volume. The electrocardiogram is usually normal, both right and left axis-deviation having been reported. Obviously conditions will change in the presence of mitral stenosis, as in Lindgren's case.[39]

The Murmur. On account of the lower pressure-gradients involved, a typical machinery murmur is not heard in every case. It is important to listen over the whole chest in a quiet room, for the murmur may be very faint. The diastolic component may be absent, but may be brought out by exercise. If present, a machinery murmur is an important physical finding, particularly in cases which do not show cyanosis or clubbing, as in our first case. If situated in the left upper lobe, it may simulate patent ductus arteriosus.

Examination of the lungs may reveal a circumscribed area of dullness, but in most cases the aneurysm is small and not situated near the surface. It may not be detected even by the

most painstaking clinical examination. The vital capacity is normal.

Splenic enlargement has been reported as slight in 2 cases.

Capillary haemangiomata of the skin and the mucous membranes are of considerable importance; their presence has been noted in many cases of pulmonary arterio-venous fistula (our case 3). The presence of haemorrhagic telangiectasis should therefore impress upon the examiner the possibility of arterio-venous fistulae in the lung. These haemangiomata are a sign of the generalized, often hereditary, vascular dysplasia. They are therefore also seen in the lining of the respiratory and gastro-intestinal tract and in the liver.[50] They are liable to rupture producing episodes of epistaxis, haemoptysis or haematemesis.

LABORATORY FINDINGS

Polycythaemia is observed with any well-established veno-arterial shunt. Haematocrit readings up to 82%, erythrocyte counts up to 11 million, and haemoglobin of 25 g.% have been reported. Polycythaemia is secondary to the anoxaemia of the arterial blood and is therefore not associated with a comparable increase in leucocytes or in platelets. A gross increase in the viscosity of the blood is caused by the polycythaemia and a marked increase in the blood volume, to well above 12 litres in many cases.

Oxygen Saturation of the Arterial Blood. As is expected, this is greatly diminished and in well-established cases may be as low as 70%. It dropped to 59% after the standard exercise-test in the case reported by Maier *et al.*[21] If the cardiac output is not increased, the blood flow through the lungs must be decreased by the amount of blood passing *via* the fistula. This may explain why the oxygen content of the arterial blood is so grossly reduced even with relatively small shunts.

Size of the Shunt. If the oxygen saturation of the systemic arterial blood and of the right ventricle are known, the exact amount of blood shunted by way of the fistula can be calculated from the following formula:

If the right ventricular output is a c.c. with an oxygen saturation of C_{rv} and if the oxygen concentration of the aortic (arterial) blood is C_{ao}, then x, the blood which is shunted through the fistula, can be calculated if we assume that the oxygen saturation in the normal pulmonary vein is 96%:

$$a \cdot C_{ao} = (a-x)96 + x \cdot C_{rv}$$
$$a \cdot C_{ao} = 96a - 96x = x \cdot C_{rv}$$
$$x = a\frac{96 - C_{ao}}{96 - C_{rv}}$$

For example, if the oxygen saturation in the right ventricle is 36% and that in the aorta 76%, the shunt x equals:

$$x = a\frac{96-76}{96-36} = \frac{a}{3}, \text{ or } 33\% \text{ of the right ventricular output.}$$

As the right ventricular output (a) can be determined by the Fick principle, both the effective pulmonary blood flow and the flow through the shunt can be calculated.

Circulation Time. The arm-lung time and the arm-tongue time are usually within normal limits, but have been reported as increased to as much as 20 or 24 seconds.[32, 33]

Radiography. The silhouette of the heart is normal. The radiological appearance of the lungs may be characteristic. A lobulated, often ill-defined shadow connected with the hilum may be demonstrated. On screening, pulsations may be visualized. If the tumour increases in size with forced inspiration against a closed glottis (Muller's test), and decreases with forced expiration (Valsalva manoeuvre) its vascular nature will be established beyond doubt. Tomography is of particular value in showing up indefinite shadows and proving their vascular origin. The pulsations of the tumour may be detected by a kymogram.

Angiocardiography. If there is any difficulty about the diagnosis and the exact localization of the fistula, angiocardiography will usually establish the diagnosis. The role of angiocardiography is well discussed by Steinberg and McClenahan.[29] The appearances vary from small pulsating subpleural tumours to a large single communication between the pulmonary artery and the pulmonary vein. In many cases one or more afferent and efferent channels are visualized, affecting not only one lobe, but often many. A selective angiocardiogram with a catheter in the artery of the affected lobe will outline the lesion more clearly than the prograde intravenous angiocardiogram.[62]

Angiocardiography is of particular value in those cases where the lesion is near the hilum and cannot be assessed from straight X-rays and screening, or in cases in which the machinery murmur may suggest a patent ductus arteriosus. The angiocardiogram may reveal additional fistulae which had remained undiscovered by other examinations, and may definitely exclude intracardiac anomalies. Angiocardiography should be carried out on all patients before operation,[33] because other associated vascular lesions have to be excluded. This was well illustrated by Jones and Thompson,[34] who reported a case with only one pulmonary vein. However, it must be stressed that angiocardiography has sometimes failed to reveal the lesion.[26] Deaths resulting from angiocardiography in cases of pulmonary arterio-venous fistula have been reported.

Nordenstrom[61] introduced a balloon-tipped catheter into the pulmonary artery, in one case *via* the femoral vein and in another *via* a cubital vein. Opaque medium injected distally into the pulmonary artery occluded by the balloon showed excellent contrast filling of the arterio-venous aneurysms in each case. There is no mention whether the cyanosis disappeared or lessened following the occlusion of the pulmonary artery or its branch. This manoeuvre is suggested as an additional diagnostic technique in difficult cases.

Cardiac Catheterization. The vascular resistance in the normal pulmonary capillary bed is said to be increased,[7] probably as the result of hypoxaemia.[8] If true, it will encourage increased flow through the fistula. This in turn will produce a further decrease in oxygen content of the arterial blood and a further increase in the peripheral resistance in the pulmonary capillaries, thus establishing a true vicious circle. We may thus explain the occasional rapid progression of the condition once cyanosis occurs.

Dye Dilution Tests. In arterio-venous fistulae of the lungs injection of Evans blue will result in two peaks of the dye dilution curve, one from the blood passing through the fistula and the other from the blood having followed the normal route through the capillary bed. Alternatively, different times of appearance with the catheter proximal or distal to the fistula may be demonstrated.[17]

COMPLICATIONS

Rupture of the vessel wall of the arterio-venous fistula either into the pleural cavity, or into the lung itself resulting in haemoptysis, is an ever-present threat and may be fatal.

Most of the complications arise from the polycythaemia. Cerebral arterial thrombosis or embolization, and fatal brain abscess have been observed,[5, 25] even in the absence of pulmonary complaints.[41, 42] Subacute bacterial endocarditis has been reported twice.[22, 21]

Cerebral manifestations of arterio-venous fistulae of the lungs are common. Dizziness, vertigo, unconsciousness, with or without convulsions, tinnitus, diplopia, headaches, aphasia, mental confusion, paraesthesiae, paralysis and weakness of the extremities, usually of the left side, having been observed by numerous authors.

Epistaxis and haematemesis from associated telangiectasis,[31] are not uncommon and may lower the red-cell count. This in turn may cause temporary disappearance of the cyanosis (our case 3).

DIFFERENTIAL DIAGNOSIS

There should be no difficulty in diagnosing an arterio-venous fistula of the lung. The first essential is a general awareness of the condition when the symptoms are those suggesting at first sight congenital heart disease, polycythaemia rubra vera, or chronic pulmonary disease.

1. *Polycythaemia Vera.* This is a common diagnosis if the patient presents for the first time in adult life and is markedly cyanosed. Thus, Goldman's case[14] had been treated with radio-active phosphorus, and the patient reported by Smith and Horton[20] had received phenyl hydrazine for long periods before they recognized the true underlying pathology. Polycythaemia vera can usually be distinguished by the presence of immature leucocytes, which are not found when the polycythaemia is of the secondary type found in pulmonary arterio-venous fistulae.

Enlargement of the spleen is not common in pulmonary arterio-venous fistulae, although it has been reported in 2 cases.

Difficulty may arise concerning the pulmonary shadows. Rounded, homogenous pulmonary shadows have been described by Hirsch[2] in polycythaemia vera which may be indistinguishable from those seen in pulmonary arterio-venous fistulae. They are probably due to infarction. However, if the lesion decreases with forced expiration, or pulsates, its true nature will not be in doubt and a machinery murmur, if heard, will be conclusive.

2. *Cyanotic Heart Disease.* Cyanosis and clubbing, when seen in the very young, immediately suggest congenital heart disease. This is usually the first diagnosis to be made, particularly if there is dyspnoea as well. Increasing dyspnoea with attacks of dizziness, convulsions and vertigo precipitated by exertion are entirely compatible with this diagnosis. However, absence of the usual physical signs should make one suspect some other cause, although it has to be kept in mind that in severe cases of Fallot's tetralogy or in cases of true- or pseudo-truncus arteriosus, very few signs may be detected clinically over the heart. The absence of abnormal cardiac signs in pulmonary arterio-venous fistulae readily excludes the more usual cyanotic congenital cardiac conditions (those associated with pulmonary or infundibular stenosis and those with severe pulmonary hypertension).[18] However, difficulty may arise with conditions giving rise to minimal cardiac signs, such as extreme tetralogy of Fallot, pulmonary atresia, some cases of Eisenmenger's complex, transposition of the great vessels and Ebstein's anomaly. However, a normal electrocardiogram and cardiac silhouette associated with an abnormal pulmonary shadow will usually settle the problem. The presence of a continuous murmur is not conclusive because it is frequently found in pulmonary atresia, being due to the greatly increased broncho-pulmonary shunts which also cause abnormal pulmonary vascular markings leading to a further point of similarity. In one of our cases, pulmonary atresia was in fact the initial diagnosis. However, pulmonary atresia can be excluded by careful auscultation,[16, 63] the presence of a sabot-shaped heart, and right ventricular hypertrophy as shown by the electrocardiogram. Angiocardiography and other special investigations will help to clinch the diagnosis.[61, 62]

3. *Bronchiectasis.* Haemoptysis, cyanosis, and clubbing, particularly in the adult, may suggest a chronic lung lesion accompanied by cyanosis. However, the history, the physical signs and special investigations will soon suggest the correct pathology. If doubt exists, an angiocardiogram may be necessary to prove that the lesion is vascular.

4. *Inflammatory or Neoplastic Lesions of the Lung.* In those cases in which cyanosis and clubbing are minimal or absent and a shadow is found in the lungs, this is the commonest diagnosis. In the young, during the pre-cyanotic stage of the condition, arterio-venous fistulae of the lung are often diagnosed as pulmonary tuberculosis, particularly if there have been attacks of haemoptysis.[57] This was the case in one of our patients, who had been hospitalized for 2 years for pulmonary tuberculosis, despite the fact that tubercle bacilli were never found in the sputum or on gastric lavage.

In the adult the question of neoplasm will arise. Careful examination of the lungs in order to detect a machinery murmur is more important in this particular type of case than any other special examination. The results of tomography may be equivocal, and angiocardiography may have to be done. The diagnosis may indeed be very difficult at a time when cyanosis, clubbing and polycythaemia have not yet developed.

COURSE AND PROGNOSIS

It is difficult to give an opinion on the prognosis in an individual case. Seaman and Goldman[4] followed up two cases for 12 and 16 years respectively, who developed no symptoms, and we have one case who has been seen for even a longer period without showing signs of deterioration. One case reported by Yater, Finnegan and Giffin[9] was 42 years of age, and had hardly any symptoms apart from an occasional mild epistaxis, and attacks of arthralgia which could not be associated with the lesion. However, on the whole, the course must be considered progressive, with enlargement of the vascular channels and degenerative changes in their walls leading to rupture and tearing into the lung.[46] We agree with Yater *et al.*[9] that adult patients with small fistulae who do not show signs of any deterioration may be kept under observation and operated upon only if changes develop.

PATHOLOGY

Essentially two types of lesions are met with:

1. A single large shunt affording a free communication between artery and vein.

2. Multiple small communications as in cirsoid aneurysm. In both forms the lower lobes are more commonly affected.

The lesion consists of tortuous, distended, thin-walled afferent arteries leading into a labyrinth of distended vessels drained by similar thin-walled efferent veins. In the course of time the vessels become more and more dilated by degeneration of some of the septa, and larger cavities are formed. They come to lie beneath the pleura and often are covered by a very pliable layer of lung tissue. As Steinberg and McClenahan[29] have pointed out, the drainage need not necessarily be directly into the pulmonary vein of the same lobe, but may be into the vein of an adjacent lobe. Although one lobe only may be mainly affected, the lesion in principle is a widespread one affecting the whole lung and sometimes even the opposite lung also.[58]

Degenerative changes take place in the lesion and a sudden increase in intrapulmonary pressure, such as may occur with coughing, may lead to rupture and haemorrhage. Depending upon whether the lesion is in the centre of the lung or near the pleura, haemorrhage will occur into the pleural cavity or into the lung tissue itself.

TREATMENT

Treatment is surgical. Surgery will be curative on removal of a single lesion in one lobe or one lung. The first case so cured was that of Hepburn and Dauphine (1942).[15] The type of treatment naturally depends on the nature and size of the lesion. If the aneurysm is small and subpleural, and can be palpated during the operation, local excision[35] may be possible. Partial resection, lobectomy,[11, 12] or pneumonectomy if fistulae are to be found in all lobes of one lung, have been carried out with great success. One case reported[59] was complicated by collateral vessels to the thoracic wall which gave rise to some difficulty.

Rienhoff[38] has shown that in the human the main pulmonary artery may be ligated without the danger of gangrene unless the venous return is obliterated as well. Thus under exceptional circumstances, ligation of the pulmonary artery feeding the aneurysms may be tried;[36,37] this was carried out in our case 2.

Packard and Waring[1] treated an arterio-venous fistula of the lung by ligation of the pulmonary artery, but suggest that the pulmonary artery should not be ligated in continuity, but should be divided. They feel that, if such treatment should not succeed, pneumonectomy is still possible.

Indications for surgery in the deeply cyanosed case with polycythaemia and haemoptysis or dyspnoea is absolute. However, in the asymptomatic cases the indications for surgery are not so clear cut. If the lesion is such that it can easily be removed by excision or lobectomy, surgery, in our opinion, is indicated in order to remove the ever-present risk of serious complications, which is greater than that of surgical removal of the fistula.[42] As in its congenital systemic counterpart, there may be multiple minute pulmonary telangiectases[19] making lung surgery impossible, and 2 cases are on record in which excision of pulmonary arterio-venous fistulae caused enlargement in other pre-existing lesions.

CASE REPORTS

Case 1

P.W., a European female aged 32, was first seen by us in 1944, at the age of 20, when she presented herself for a Baker's cyst of the right knee. She gave a history of having spent 2 years in hospital for a tuberculous infection of the lung between the ages of 10 and 12; otherwise she had been perfectly well. She was never cyanosed or dyspnoeic and had no clubbing.

On routine general examination, a continuous murmur was heard over the right middle zone of the chest behind. Otherwise the examination of all the systems was completely normal. There was no polycythaemia nor familial history of telangiectasia, bleeding or cyanosis. Electrocardiogram normal. Blood pressure 120/70 mm. Hg. Blood count normal. The clinical diagnosis was arterio-venous fistula of the lung. Examination of the original X-ray taken 10 years previously, as well as subsequent yearly pictures, showed that the lesion had remained the same throughout the years and that the original picture was compatible with the present diagnosis (Fig. 1).

The patient remained perfectly well for the next 10 years, and went through two normal pregnancies without showing any signs of cyanosis, dyspnoea or clubbing. The blood counts were repeatedly normal. In 1954 an angiocardiogram was done. The abnormal shadows filled immediately with dye and individual abnormal vessels could be clearly outlined in the right lung without, however demonstrating their exact course (Fig. 2). There was rapid filling of the left heart. The appearances were those of an arterio-venous fistula in the right middle lobe (Figs. 2-3). It was difficult to be certain that the vascular pattern in the remaining lung was normal.

In view of the fact that the clinical picture had been the same for 20 years, surgery was not recommended.

In 1956, the patient was again seen and was in perfect health.

Case 2

E.E.B., a European male aged 23, was first admitted to Groote Schuur Hospital in July 1952. He gave a history of intense cyanosis and clubbing since birth. As long as he could remember he had

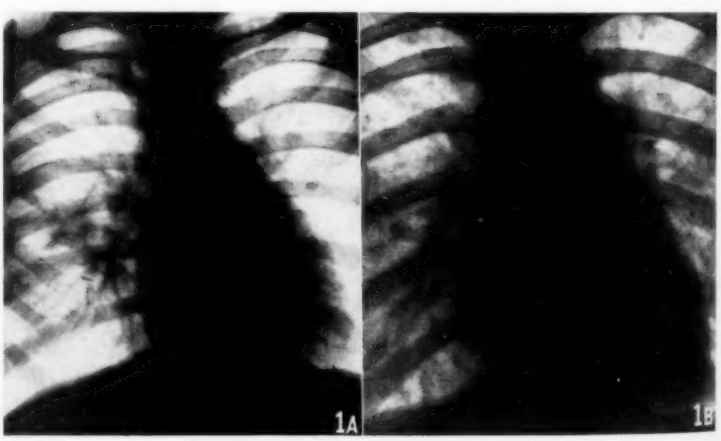


Fig. 1. Case of pulmonary arterio-venous fistula which has been under observation for 23 years, but was originally diagnosed as pulmonary tuberculosis (Case 1). Teleradiogram of chest: A, 1934: B, 1944.
There has been no deterioration of the condition.

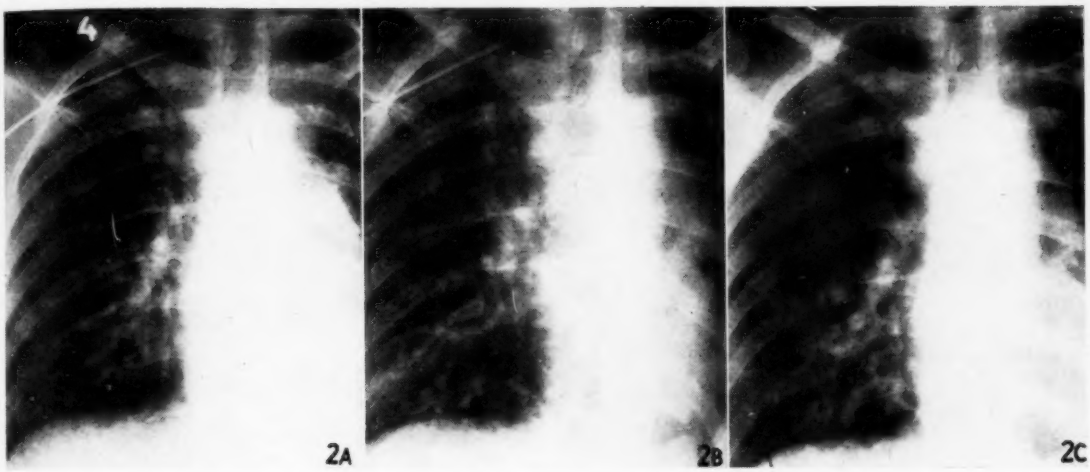


Fig. 2. Angiocardiograms of same case as Fig. 1, showing arterio-venous fistula in right middle lobe. The pictures taken at 1 second intervals (Case 1).

been dyspnoeic and was never able to participate in school sports. At the time of admission. he could only walk about ¼ mile on the level at an ordinary pace, and then, because of dyspnoea and palpitations, he had to rest. He never squatted. There was an episode of haemoptysis several years previously and again 3 weeks before admission.

On Examination: Marked central cyanosis and intense polycythemia with grade-III clubbing of toes and fingers. Blood pressure 95/45 mm. Hg. Apex beat in the 5th intercostal space ¼ inch beyond mid-clavicular line. Visible and palpable pulsation present in the 2nd left interspace. Basal second sound loud and just split at the pulmonary area. Soft grade-II systolic murmur at apex. No continuous murmur over heart or lung fields; no telangiectases; no other abnormal physical signs.

On screening, the heart appeared normal in size with prominent and pulsating main pulmonary vessels. On X-ray, the heart size was normal. The hilar shadows were abnormally prominent on the right (Fig. 4).

The electrocardiogram was normal.

Blood Examination: Haemoglobin 18·5 g.%. Packed cell volume 87%. Leucocytes 10,700 per c.mm.

Angiocardiography (13 August 1952) presented some interesting and puzzling features. There was a distinct narrowing of the superior vena cava as it entered an extremely small right auricle, with a hold-up of the dye. In the early pictures a large lobulated mass is outlined, which is drained by a large abnormal vein entering the left auricle (Fig. 5). The oblique views showed the mass well filled with dye lying behind and below the left auricle in front of the vertebral column, before any visible filling of the pulmonary arteries (Fig. 6).

As he refused surgery he was discharged, only to be readmitted 8 weeks later after a severe haemoptysis. He had now developed slight ankle oedema and a large indolent weeping ulcer on the left ankle.

On this admission a bronchogram was done and found to be normal. He improved on bed rest and was discharged after 4

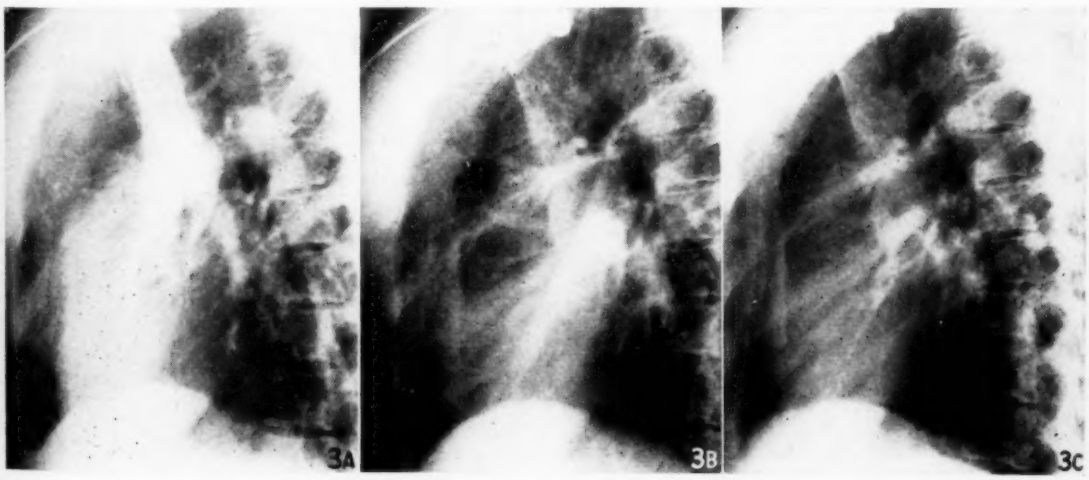


Fig. 3. Angiocardiogram right posterior oblique projection, same case as Figs. 1 and 2. Note the vascular markings in the right middle lobe with early filling of left auricle in Fig. 3B (Case 1).

weeks to await surgery. On 18 March 1955 the patient was readmitted as an emergency case after a severe haemoptysis 24 hours before admission. After repeated blood transfusions, a right-sided thoracotomy was performed. A large vascular sac lying superior to the inferior vena cava and slightly posterior to the pulmonary veins was displayed. The inferior vena cava appeared to be entering the sac and not the right auricle. Great difficulty was experienced in exploring the right pulmonary artery; this was finally ligatured proximal to the presumed origin of the fistula, without excising the vascular tumour or any lung tissue. The right auricle was small, with an auricular appendage less than 2 cm. in diameter.

Subsequent Course: The patient made an uneventful recovery but did not show any alleviation of the signs or symptoms.

Comment: This case of complicated arterio-venous fistula of the lung presented from birth with intense cyanosis, clubbing, dyspnoea and later developed repeated haemoptysis. Only a soft grade-II systolic murmur was heard. Ligation of the right pulmonary artery, although not producing any improvement of the condition, was not followed by any complications.

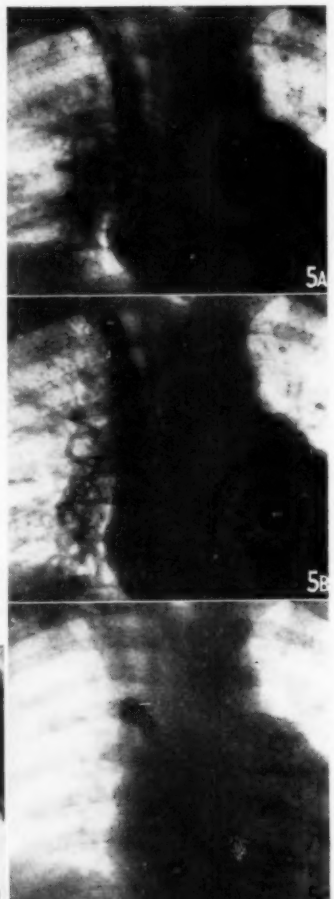


Fig. 4. Teleradiogram of case 2. Note the vascular markings at the right lower lobe.

Fig. 5. Angiocardiogram of same case as Fig. 4, showing in B and C typical arterio-venous fistula drained by at least one large vessel entering the left auricle (C) case 2.

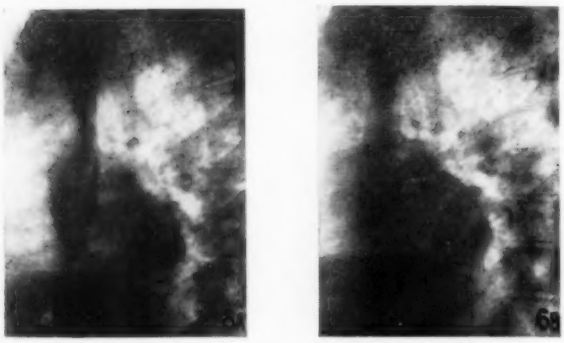


Fig. 6. Right posterior oblique projection of angiocardiogram (case 2). Note pooling of dye in large saccular dilatations before there is filling of the remainder of the lungs.

Case 3

M.W., a European female aged 45 years, had for the last 25 years noticed discoloured (bluish), slightly elevated marks on her tongue. For 25 years she had bled intermittently from the nose. Two years before admission she bled so profusely that she had to be transfused. Originally the nose bleeding tended to occur pre-menstrually, but for the last 15 years bleeding occurred at any time. For 2 months before admission there had been progressive dyspnoea.

Family History. There is a definite history of hereditary haemorrhagic telangiectases (Fig. 7).

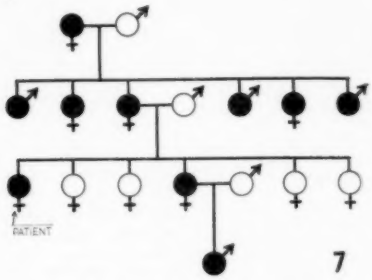


Fig. 7. Family tree of case 3. The shaded symbols indicate members of the family who are afflicted with teleangiectasis.

On Examination (21 May 1956) there was marked pallor and dyspnoea from the post-haemorrhagic anaemia, moderate clubbing and telangiectases on her fingers, lips, tongue and palate (Fig. 8). A grade-III systolic murmur was audible at, and just within, the apex. A soft continuous murmur was noted at the base of the left lung posteriorly. Blood pressure 140/60 mm. Hg. Packed cell volume 31%.

In view of the packed cell volume of 31% and haemoglobin of 5 g.%, the patient was transfused; after the anaemia had been corrected, central cyanosis appeared. Electrocardiogram was normal. The X-ray revealed numerous shadows compatible with arterio-venous fistulae in the left lower lobe (Figs. 9 and 10). The heart was not enlarged. Tomography of the remaining lung was clear.

The patient did not submit to any special investigations.

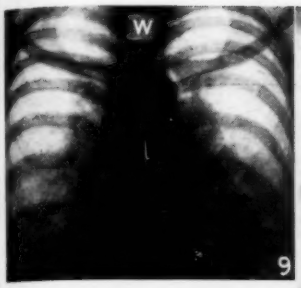
Comment. The interest of this case lies in the association of hereditary multiple telangiectasia and pulmonary arterio-venous aneurysm. There was a continuous murmur, with clubbing and cyanosis. When first seen she was not cyanosed because of the anaemia. The presence of clubbing in a patient with hereditary telangiectasia suggested pulmonary arterio-venous fistula even though she was not cyanosed. X-rays confirmed this. After transfusion and restoration of normal haemoglobin deep central cyanosis became obvious.

Case 4

S.S., a European female aged 10, was first seen in November 1954. She had been blue from birth. She was of normal intelligence and physical development. She had always been tired, and dyspnoeic on much effort. This was relieved by squatting. She had always had a tendency to productive cough, but there was never haemoptysis. At school she did not participate in games. There was no history of fainting, but she experienced central, stabbing chest pain on exertion.

Her 8 sisters and brothers are well. She has a cousin with congenitally absent upper limbs.

On Examination: A well-built, tall girl exhibiting central cyanosis of moderate intensity, moderate clubbing of fingers and toes; arachnodactyly and high arched palate. No. angiomata



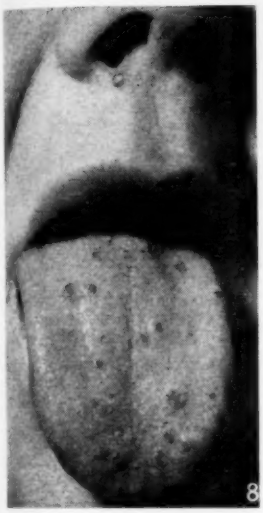


Fig. 8. Haemorrhagic telangiectases affecting the skin and the tongue of case 3.
Fig. 9. Teleradiogram of case 3, showing vascular shadows in left lower lobe.
Fig. 10. Tomocardiogram of case 3, showing large shadow in left mid zone.

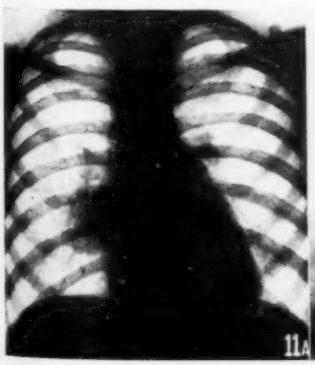
on the face or mucous membranes. Blood pressure 95/66 mm. Hg. On palpation, moderate right ventricular lift and pulmonary-artery pulsation felt. Apex beat in 4th interspace in mid-clavicular line, normal left ventricular in type.

On auscultation a very loud, early pulmonic systolic click present at 4th intercostal space and at pulmonary area, with a closely split pulmonary second sound. A distinct systolic murmur was present posteriorly on the right side medial to the right scapula.

Electrocardiogram. Evidence of slight right auricular hypertrophy; inversion of T waves in V1 and V2. Within normal limits for the age.

Haemoglobin 20 g.%. Packed cell volume 69%.

X-rays. On screening there was enlargement of the pulmonary artery and an obvious pulsating shadow at the right hilum, but unlike a large right pulmonary artery. Right ventricle not enlarged. The X-ray picture (Fig. 11a) show a well demarcated rounded shadow at and below the right hilum, with some increase in vascular markings in the right lower lobe. In the lateral projection a saccular lobulated shadow was seen below the right main bronchus (Fig. 11).



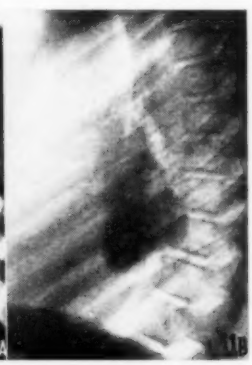


Fig. 11. Teleradiogram showing large globular mass below right hilum (case 4). A, anterior posterior projection; B, lateral view.

On tomography there was a definite prominence of the pulmonary vessels at the right hilum, and a globular shadow was seen behind the right lower border of the heart. This suggested a highly vascular mass.

The prograde angiocardiogram (Fig. 12) showed the markedly dilated main right pulmonary artery crossing the mid-line with marked 'pruning' of the small pulmonary vessels as is usually seen in pulmonary hypertension. The shadow in the right lung-field filled immediately from the right main pulmonary artery. The left auricle was already opacified after 3 seconds, indicating an extremely fast flow from the right to left through an arterio-venous fistula. Judged by the opacification of lung fields there was little flow of dye through the periphery of the right lung as compared with the left. No other abnormality was seen.

Cardiac catheterization revealed the following data: Main pulmonary-artery pressure 18/5 mm. Hg. Right pulmonary-artery pressure 16/4 mm. Hg. Pulmonary capillary venous pressure zero. Right ventricular pressure 24/1 mm. Hg. Right auricle 2 mm. Hg.

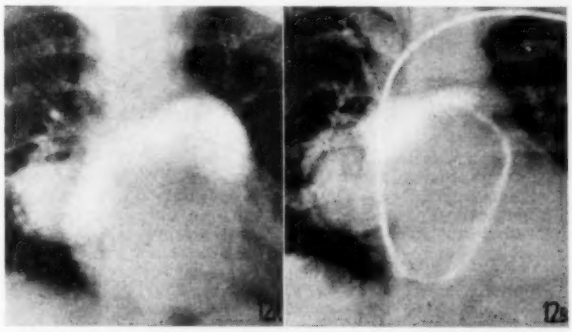


Fig. 12. A. Prograde angiocardiogram showing immediate filling of a large saccular mass. B. Selective angiocardiography reveals the connection with the right pulmonary artery (case 4).

These normal pressures (no pulmonary hypertension) excluded the possibility of a right-to-left shunt occurring through a ductus or a ventricular septal defect—nor is there any evidence of an atrial septal defect. There must therefore be a considerable shunt of pulmonary-artery blood into a pulmonary vein *via* a pulmonary arterio-venous fistula to account for the central cyanosis. A low diastolic pressure, indicating a reduced pulmonary vascular resistance, provided further evidence for this diagnosis.

Operation was performed on 16 February 1955 by Mr. W. L. Phillips. Through a standard right posterio-lateral thoracotomy incision at the right 6th intercostal space, a fistulous 'lake', partly in the right lower lobe and partly extra-pulmonary was noted. One major communication between the pulmonary artery and pulmonary vein was exposed and ligated. An abnormal entry of pulmonary veins into the right auricle was seen. A right lower lobectomy was performed.

Pathologist's Report. The operation specimen consisted of right lower lobe of lung, weight 85 g. There is a marked dilatation of the pulmonary hilar vessels and prominence of pleural lymphatics. No naked-eye evidence of arterio-venous fistula was seen. Histologically, the large vascular channels are larger and more numerous than usual; none of these vessels or the smaller ones show abnormal structural features (Fig. 13).

The patient had a very stormy post-operative course and died 3 days after operation from peripheral collapse.

At Autopsy: No adequate cause for death was found. There was gross congestion of all the organs and vessels. No evidence of any vascular abnormality was found in the remaining lung, or elsewhere. Heart weight 147 g. The left ventricle wall measured

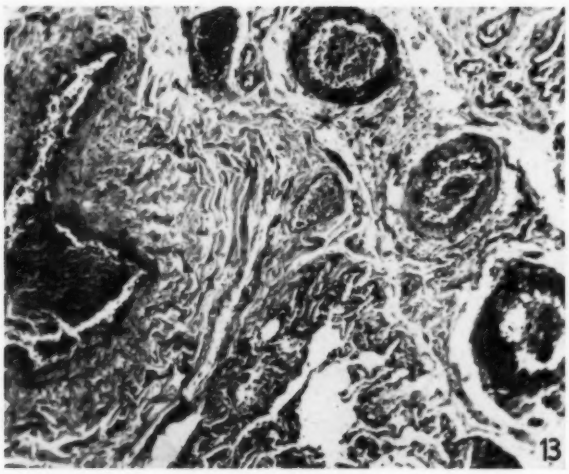


Fig. 13. Microphotograph showing the large abnormal channels in the lung tissue removed at operation (case 4).

0·8 cm. in thickness and the right ventricular wall 0·3 cm. Neither chamber appeared hypertrophied or dilated. No abnormal communication between chambers. Valves normal.

Comment: This patient presented, from birth, dyspnoea on effort and squatted. She never had any haemoptysis. There was a systolic murmur and a pulmonary ejection sound only.

Case 5

A.C., a European female aged 22, was first seen with myxoedema in 1943. No abnormalities were then noted in the heart or lungs. The electrocardiogram showed the low voltage of myxoedema, and on X-ray there was slight generalized cardiac enlargement. Haemoglobin 88 g.%. Red cells 3,500,000 per c.mm. The patient responded well to thyroid therapy.

Three years later (September 1946) she was readmitted with a sore throat, for which she had been given sulphadiazine. This was followed shortly afterwards by swelling of the legs and a purpuric eruption. On examination she had the characteristic features associated with thrombocytopenic purpura with anaemia and splenomegaly.

Examination revealed for the first time the presence of a continuous murmur, best heard at the base of the right lung posteriorly. Two weeks later the patient developed a cough and frequent haemoptyses. She was X-rayed and diagnosed as pneumonia (Fig. 14).

After her discharge she remained well until 31 January 1947, when she developed a cold and generalized constitutional symptoms. This was followed by a purpuric rash and polyarthritis. A blowing systolic murmur was heard over the left anterior chest wall. This changed into a continuous murmur at the left base, best heard posteriorly radiating into the left axilla. There was palpable pulsation over the pulmonary artery. The splenomegaly was still present. Blood pressure normal.

The blood contained 4,000,000 red cells per c.mm., and haemoglobin 12 g.%. The electrocardiogram showed right axis deviation in the standard leads. On X-ray the main, right and left pulmonary arteries were prominent. Pulsation was most marked in the main pulmonary arteries and at the right border of the heart. There was no chamber enlargement. The left hilum was elevated. The appearances were those associated with congenital heart disease, with a left-to-right shunt. Bronchography normal. The diagnosis of Henoch-Schonlein purpura was made to account for the anaemia, rash and polyarthritis. During the stay in hospital the rash and arthritis settled and the patient was discharged fit. She was readmitted as an emergency case in 1948 in severe congestive heart failure and pulmonary infarction. She retrogressed rapidly and died in spite of treatment.

Post-mortem examination revealed the following: (1) Bilateral arterio-venous aneurysms of lungs. (2) Multiple pulmonary emboli. (3) Right ventricular hypertrophy. (4) Dilatation and atheroma of pulmonary arteries. (5) Chronic passive congestion in liver and spleen. (6) Ascites.

The heart weighed 418 g. There was marked hypertrophy of the right ventricle with a dilated pulmonary artery showing patches of atheroma.

Both lungs showed chronic passive congestion. The right lung weighed 240 g., the left 225 g. Both lungs were small and partially collapsed. In both lungs arterial thrombi occluded the lower branches of the pulmonary artery. A small arterio-venous fistula

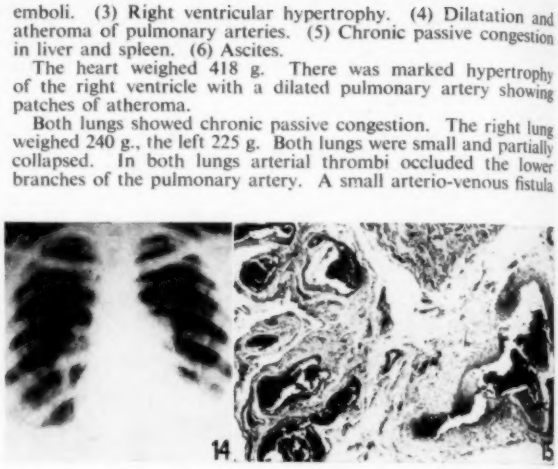


Fig. 14. Tomocardiogram of case 5.
Fig. 15. Arterio-venous fistula removed at post-mortem (case 5).

was present in the left apex and a large one in the right lower lobe (Fig. 15). On histological examination the lungs showed infarction and fibrosis with very marked endarteritis of the arteries. There were organizing thrombi in the bigger arteries with large areas of lung infarction.

The liver showed marked congestion of the sinusoids but not of the central vein.

DISCUSSION

The great interest of these five cases lies in the variety of their presentation.

Whereas case 2 exhibited the full-blown clinical picture of central cyanosis, clubbing, dyspnoea and haemoptysis, case 1 has remained completely asymptomatic for 20 years. In case 3 the patient presented with recurrent epistaxis from hereditary telangiectases. She had dyspnoea but was not clinically cyanosed, though she showed moderate clubbing. The patient in case 4 never had haemoptysis, but showed cyanosis and clubbing, was breathless, and squatted. In case 5 the patient had haemoptysis but showed no clubbing, cyanosis or dyspnoea and the diagnosis was never entertained during life.

The difficulty in diagnosing the condition in cases which do not show cyanosis and clubbing is well illustrated in case 1, where the patient had been treated for 2 years in an institution for tuberculosis of the lungs. On the other hand, when cyanosis, clubbing and polycythaemia are present the picture of congenital heart disease may be so perfectly simulated that the condition is first diagnosed as such.

Pulmonary atresia may present particular difficulty in differential diagnosis, because it is associated with a continuous murmur and cyanosis. The presence of an added sound in early systole in this condition—the aortic ejection sound—is not heard in pulmonary arterio-venous aneurysm. There may be no murmurs in pulmonary atresia and the added sound may be the only abnormality. A pulmonary ejection sound was heard in one of our patients (case 3). The continuous murmur of the patent ductus or bronchial collaterals associated with a cyanotic congenital heart condition such as Fallot's tetralogy or pulmonary atresia

ought not to present much difficulty in differential diagnosis because of the presence of the associated cardiac defects.

Case 3 when first seen had anaemia and only showed cyanosis when the blood count was restored. The combination of clubbing and anaemia with telangiectasia suggested the diagnosis of pulmonary arterio-venous aneurysm in preference to congenital heart disease.

Bleeding from the arterio-venous fistula is the most serious of the complications; it may take place into the pleural space or present as haemoptysis. It occurred in two of our cases. Haemoptysis calls for surgical intervention, necessitating exact anatomical localization of the lesion. This is not always possible by clinical means.

Only 3 of our cases presented with a typical machinery murmur. The remaining two had a systolic mumur only.

Straight X-rays will help to localize the lesion, but X-ray appearances vary greatly, from large globular and haemangiomatous masses to some slight increase in vascular markings. Cases have been described with negative straight X-rays so that oblique pictures, tomography and angiocardiography may have to be done. Four of our cases had abnormal shadows on X-ray.

It is not only important to know which part of the lung is affected but even more important whether the remaining lungs are clear. Indeed, removal of one lobe or an entire lung containing a well recognised fistula has made matters worse when the other lung has also been affected by a similar lesion. Angiocardiography is, therefore, indicated in the diagnosis of these cases if surgery is contemplated.

The timing of the operation is not usually difficult and is indicated if the patient has haemoptysis or shows the full blown picture, but is probably unnecessary in the asymptomatic case. Few cases have been followed up for long enough to justify a different approach. Case 1, is therefore, of considerable interest as data, including X-rays, are available for 23 years, and the condition has been recognised as an arterio-venous fistula for 12 years. This case shows clearly that in the asymptomatic case the lesion does not necessarily progress.

Whereas lobectomy or pneumonectomy is the treatment of choice, occasionally it may not be possible to carry out segmental resection. Ligation of the artery has then been performed. No complication arose from ligation of one of the main branches of the right pulmonary artery (case 2).

The electrocardiogram was normal in all our cases as is usual in this condition.

In contrast to its systemic counterpart, pulmonary arterio-venous fistula does not usually effect the heart and does not usually produce congestive cardiac failure. In only one of our patients was the heart enlarged, and in this case there were pulmonary emboli.

We would like to thank the physicians of Groote Schuur Hospital for their kindness in allowing us to investigate their cases and to our colleagues in private practice who referred their patients to us.

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A DISEASE RESEMBLING POLIOMYELITIS

REPORT OF AN OUTBREAK IN JOHANNESBURG

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During the last epidemic of poliomyelitis in Johannesburg, which lasted from August 1954 to March 1955, frequent atypical cases presented themselves at the Fever Hospital in Johannesburg with symptoms not quite characteristic of true poliomyelitis and in which on subsequent investigation the diagnosis of poliomyelitis could not be made. We report 14 such cases. The disease presented in both males and females. The youngest was 8 years of age, and the oldest was 47. The greatest frequency was in the second and third decade. Females (10) were affected more frequently than males (4). In the same period there were 200 cases of poliomyelitis, proved by virus studies.

The details of our cases are given below. The most significant features in all of them were as follows:

Type of Onset. In most cases symptoms appeared fairly abruptly, but in one or two, e.g. cases 4 and 5, there were symptoms of a slower onset. In cases 4 and 5 the patient felt ill 3 weeks before acute symptoms occurred. In case 1 there was a history of abdominal pains 2 weeks before the more severe symptoms appeared. In case 10 there was a history of coryza 2 weeks before admission. All cases presented with severe headaches, mainly over the frontal region. Associated with headache there was frequently severe stiffness of neck and back. In some cases back stiffness was more prominent than neck stiffness. Pyrexia was either absent or did not exceed 100°F except in cases 3 and 9.

Motor Symptoms and Signs. These varied from mild weakness to actual paralysis. In case 8 there was weakness of both legs. There was often weakness of single muscles, e.g. biceps, deltoid or quadriceps; that is to say, almost invariably proximal muscular weakness. Facial weakness occurred fairly frequently. At no time were abnormalities of the bladder, bowel or diaphragm observed.

Sensory Symptoms and Signs. Muscle pain was an outstanding and obvious feature in this series. The pain was deep-seated in the muscles involved and the muscles were sensitive to touch. In some cases pain was so severe that pethidine had to be administered. As described above, stiff painful neck and backache were noticeable features. A history of paraesthesiae was occasionally obtained. No objective neurological sensory signs were present. Apart from frequent facial-nerve involvement, no other cranial nerve palsies were observed.

Clinical Investigations. The blood count was normal. There was no evidence of any white-cell disturbance. The blood sedimentation rate was normal or only slightly raised. The cerebrospinal fluid was normal in pressure, and its protein content was almost invariably below 50 mg.% which is taken as the upper limit of normal in Johannesburg. In all cases the following tests proved negative: (1) Examination of the stools for poliomyelitis and Coxsackie virus, and (2) of the blood for virus agglutination and Rickettsia, and (3) the Paul-Bunnell test (reversible).

Sequelae. In 3 cases sequelae occurred. Case 14 suffered a relapse within 2 weeks after discharge from hospital; all muscles previously involved were again affected; the left deltoid, quadriceps and tibialis anticus were weak. Case 4 had a stiff back at the time of discharge. No mental symptoms were observed after discharge in any case.

CASE REPORTS

Case 1. *J.E.L. Female. Aged* 15½. *Schoolgirl.*

Admitted 29 December 1954. Discharged 7 January 1955. On the day of admission the patient developed severe frontal headache and pain in her back and neck. The next day she noticed that her left leg was weak. Two weeks previously she had had an attack of acute upper abdominal pain, which lasted for 3 days and cleared up spontaneously.

Examination. The patient was a well built female. Temperature 100·6°F. Pulse rate 110 per minute. No neck stiffness but considerable back stiffness. Slight weakness of quadriceps and hamstrings on the left. Absent knee jerk on the left.

Progress. The temperature settled after 24 hours. Weakness and

reflex change disappeared in 24 hours. The back stiffness, however, persisted for 6 days. The patient was discharged well.

Investigations. (1) WBC 6,000 per c. mm. ESR 8 mm. in 1 hour. (2) CSF: 1 lymphocyte and 2 erythrocytes per c. mm.; 26 mg.% of protein. (3) Stools: No poliomyelitis virus; no Coxsackie virus.

Case 2. *M.P. Aged* 16. *Female. Clerk.*

Admitted 17 February 1955. Discharged 8 March 1955. This patient had a severe attack of 'flu' 2 weeks before her present illness. The history of her present complaint was of 3 days' duration, starting with severe lower-back pain. She continued working, however. On the day before admission she complained of frontal headache and vomiting. She was then seen by her general practitioner, who advised admission to hospital as a case of poliomyelitis.

Examination. Apyrexial at the time of admission. Stiff neck and back. Positive Kernig's sign. Generalized weakness of upper and lower limbs bilaterally. Reflexes were all depressed.

Progress. Power returned to normal in 4 days. Reflexes, however, were still depressed at this time, and only returned to normal on the 10th day. She was discharged with no weakness or spasm.

Investigations. (1) WBC 9,000 per c. mm. ESR 9 mm. in 1 hour. (2) Lumbar puncture (17 February). 5 lymphocytes per c. mm. and 29 mg.% of total protein; (28 February) no cells and total protein 34 mg.%. (3) Stools: No poliomyelitis virus; no Coxsackie virus.

Case 3. *M.T. Aged* 26. *Nursing Assistant.*

Admitted 24 February 1955. Discharged 2 March 1955. This patient was perfectly well until the day before admission, when she developed severe frontal headache. On the evening before her admission to hospital she noticed that her back was stiff and painful.

Examination. Temperature 102°F. This settled within 24 hours. On admission the patient looked ill and lethargic. She had considerable pain in her back, a very stiff neck and back, weakness of her left deltoid, and weakness of flexion of her left elbow. There were no sensory changes. Reflexes of the left arm were depressed. Knee and ankle jerks on both sides were depressed.

Progress. The stiffness of the neck and back cleared completely in 5 days. The weakness, too, was ephemeral, disappearing within 36 hours.

Treatment. Pethidine was given to control the pain in the back.

Investigations. (1) WBC 8,800 per c. mm. ESR 13 mm. in 1 hour. (2) Lumbar puncture: 1 lymphocyte per c. mm.; 31 mg.% of total protein. (3) Stools: No poliomyelitis virus; no Coxsackie virus.

Case 4. *W.L. Aged* 16. *Schoolgirl.*

Admitted 7 March 1955. Discharged 22 March 1955. This patient had been ill for 3 weeks before admission. During this time she was confined to bed for 5 days with a persistent cold. For 4 days before admission she had severe headache and cramps in her left leg. At school on the day before admission she noted that she could not move her left leg. At the same time her headache increased in intensity and was occipital and frontal in situation.

Examination. Apyrexial. Very stiff neck and back. Weak deltoid on the right. Weak extension and flexion of right elbow. Weakness of whole of leg on the left side. Hypoaesthesia of left leg. Reflexes of right arm depressed. Reflexes of left lower limb slightly increased.

Progress. In 2 days muscular power returned in the affected limbs. By this time all the reflexes were back to normal, completely returning in approximately 6 days.

Investigations. (1) WBC 7,300 per c. mm. ESR 14 mm. in 1 hour. (2) Lumbar puncture: 1 polymorph per c. mm.; 26 mg.% of total protein. (3) Stools: No poliomyelitis virus; no Coxsackie virus.

Case 5. *C.P. Schoolgirl. Aged* 11½.

Admitted 18 February 1955. Discharged 22 February 1955. Patient had been ill with 'flu' for 3 weeks before her admission. On the day of admission she developed headache, together with pain in the neck and lower back. She also developed muscular pains and tenderness involving all four limbs.

Examination. Temperature 100°F. This settled within 24 hours. Marked back and neck stiffness present. Seventh-nerve palsy of the right face. Generalized muscular weakness mainly affecting the deltoid muscles in the upper limbs and the quadriceps muscles in the lower limbs. These muscles were found to be tender to palpation. There were no changes evident in the sensory system. Reflexes were all originally depressed.

Progress. The illness was characterized by very rapid improvement in all the signs and symptoms.

Investigations. (1) WBC 10,000 per c. mm. ESR 5 mm. in 1 hour. (2) Lumbar puncture: No cells; 22 mg.% of total protein. (3) Stools: No poliomyelitis virus; no Coxsackie virus.

Case 6. *J.C.S. Aged* 23. *Male. Wireless Licence Inspector.*

Admitted 23 March 1955. Discharged 8 April 1955. This patient had been feeling off colour for 5 days before admission. Three days before admission he developed headache, stiff neck and pain in the lower back. He went to bed after the onset of these symptoms, and on 23 March they had become very much worse and admission to hospital was advised by his general practitioner, who had diagnosed poliomyelitis.

Examination. Complaining of considerable backache. Apyrexial on admission. Stiffness of neck and back present. Seventh-nerve palsy on the right. Weakness of quadriceps on both sides. Reflexes were all present and equal. No impairment of sensation.

Progress. After 3 days he ran a high temperature. On examination it was found that although the weakness in his lower limbs had improved, the biceps and triceps muscles on both sides were weak. Within a week, however, all his weakness had cleared and he was no longer complaining of pain and stiffness in his back.

Treatment. The pain responded to aspirin, phenacetin and codeine tablets.

Investigations. (1) WBC 11,800 per c. mm. ESR 1 mm. in 1 hour. (2) Lumbar puncture: 1 polymorph and 3 lymphocytes per c. mm.; 34 mg.% of total protein. (3) Stools: No poliomyelitis virus; no Coxsackie virus.

Case 7. *H.J. Aged* 24 *years. Nursing Aid.*

Admitted 8 April 1955. Discharged 12 April 1955. This patient's main complaints were headache and stiffness of the neck and back, which had been present for 4 days before admission.

Examination. Apyrexial on admission and remained so. Marked neck and back stiffness. There was a generalized muscle weakness. Tone was normal. All reflexes were present and equal.

Progress. The pain and stiffness persisted for 2 days and then cleared up spontaneously, leaving no residue.

Investigations: (1) WBC 8,400 per c. mm. ESR 3 mm. in 1 hour. (2) Lumbar puncture: 1 polymorph and 1 lymphocyte in 1 c. mm.; 51 mg.% of total protein. (3) Stools: No poliomyelitis or Coxsackie virus.

Case 8. *O.S. Aged* 6 *years. Schoolboy.*

Admitted 18 July 1954 and discharged 31 August 1954 (not during the poliomyelitis epidemic. This boy had 'flu' 2 weeks before admission. Six days before admission he complained of pain in his legs. This was followed by difficulty in walking. Two days before admission he started complaining of pain in his arms.

Examination. Apyrexial. Moderate neck stiffness and marked back stiffness. Cranial nerves intact. Motor power appeared unimpaired. All reflexes were present and equal. Sensation was normal.

Progress. After the patient had been in hospital for 3 days, it was found that both biceps and ankle jerks were diminished. At this time the power of the child's grip had diminished considerably. He had also developed weakness of dorsiflexion of both feet. There was no rise in temperature, white-cell count, or ESR coincident with the spread of his weakness. Power began to return in 4 days and was completely restored in 1 week, although back stiffness persisted for nearly 2 weeks. On discharge his quadriceps were still weak.

Investigation. (1) WBC 10,000 per c. mm. ESR 5 mm. in 1 hour. (2) Lumbar puncture: 1 lymphocyte per c. mm.; 44 mg.% of total protein. (3) Stools: No poliomyelitis or Coxsackie virus.

Case 9. *D.P. Aged* 16. *Schoolgirl.*

Admitted 17 February 1955. Discharged 27 February 1955. Sudden onset of severe headache and backache 2 days before admission. On the day of admission the backache was very much worse and it was noticed that her back was very stiff.

Examination. Temperature 102·2°F, which settled within 24

hours. There was slight neck stiffness and considerable back stiffness. No impairment of motor power could be found. Sensation was normal. The triceps reflexes were bilaterally diminished but returned to normal within 2 days.

Progress. The neck and back stiffness cleared up in 2 days. It was then found that the left knee jerk was depressed. The patient was discharged with no residual signs.

Investigations. (1) WBC 4,500 per c. mm. ESR 6 mm. in 1 hour. (2) Lumbar puncture: 3 lymphocytes per c. mm.; 21 mg.% of total protein. (3) Stools: Negative for poliomyelitis and Coxsackie virus.

Case 10. *E.M. Aged* 36. *Female. Shop Assistant.*

Admitted 23 April 1955. Discharged 26 April 1955. She had a history of feeling lame in both legs and backache for 2 weeks. These symptoms persisted until 2 days before admission when they became very much worse, and headache was noted. She complained of a tingling sensation in the hands on the day before admission.

Examination. Apyrexial. Slight neck stiffness. Marked back stiffness. Right seventh-nerve palsy. No other motor weakness present. No abnormality in sensory system was detected. All reflexes were depressed.

Progress. The facial-nerve palsy cleared in two days. By that time the neck and back stiffness had cleared as well.

Investigations. (1) WBC 12,200 per c. mm. ESR 2 mm. in 1 hour. (2) Lumbar puncture: 1 lymphocyte per c. mm. 40 mg.% of total protein. (3) Stools: No poliomyelitis or Coxsackie virus.

Case 11. *W.B. Aged* 22. *Female. Bookkeeper.*

Admitted 27 February 1955. Discharged 8 March 1955. Admitted with 5 days' history of lethargy, worse during the last 3 days, when she became pyrexial and started complaining of headache and pain and stiffness of neck and back.

Examination. Apyrexial. In considerable discomfort because of back pain and stiffness. Considerable neck stiffness present. Back was stiff and painful on examination. Weakness of both deltoid muscles present. All other reflexes were diminished.

Progress. The headache persisted for 3 days after admission. Power returned to the deltoid muscles within 4 days and the back stiffness had disappeared after 1 week.

Investigations. (1) WBC 4,500 per c. mm. ESR 16 mm. in 1 hour. (2) Lumbar puncture: No cells. Total protein 21 mg.%. (3) Stools: No poliomyelitis or Coxsackie virus found.

Case 12. *O.O. Aged* 8. *Schoolboy.*

Admitted 3 May 1955. Discharged 12 July 1955. Three days' history of headache, which became very much worse on the day before admission.

Examination Apyrexial. Slight neck stiffness; marked back stiffness. Cranial nerves intact. Motor system normal. No sensory disturbance. All reflexes were present except for the right biceps jerk which, however, returned to normal within 4 days.

Progress. The neck and back stiffness were persistent and were still present after a month of physiotherapy and hot packs. These signs, however, cleared up before the patient was discharged.

Investigations: (1) WBC 1,900 per c. mm. ESR 7 mm. in 1 hour. (2) Lumbar puncture: 33 RBCs per c. mm. 21 mg.% of protein. (3) Stools: No poliomyelitis or Coxsackie virus.

Case 13. *F.M. Aged* 33. *Male. Clerk.*

Admitted 4 March 1955. Discharged on 27 March 1955. Two days' history of headache and generalized body pains.

Examination. An apathetic, obese male. Apyrexial. Stiffness of neck present. Back was stiff and painful. Cranial nerves intact. Marked weakness of left upper limb, particularly left deltoid. Lower limb on the left was very weak as well. The muscles maximally involved where the hamstrings, quadriceps and tibialis anterior. All reflexes on the left half of the body were diminished in intensity.

Progress. Pain was considerable and the patient had to be given pethidine tablets. After a week he was feeling much better, although he still had some weakness of the left deltoid and the left quadriceps. His back, too, still had some stiffness. He was then allowed up, but after a day he complained of more back pain and stiffness and pain in his limbs and he had to be returned to bed. Four days after this episode he was very much better. All his muscle power had returned. His reflexes were now all present and equal, but slightly diminished generally. During his stay in hospital he was a most depressed and apathetic individual. He was discharged from hospital on 27 March feeling quite well. Within 2 weeks of discharge his weakness had returned. He was admitted to a general medical ward for investigation and the diagnosis of Icelandic disease was again made.

Investigations. (1) WBC 11,600 per c. mm. ESR 4 mm. in 1 hour. (2) Lumbar puncture: No cells. 45 mg.% of total protein. (3) Stools: No poliomyelitis or Coxsackie virus found. (4) Muscle biopsy on 10 March—normal muscle.

Case 14. *R.M. Aged* 47. *Housewife. Female.*

Admitted 7 March 1955. Discharged 20 March 1955. Three weeks before admission the patient noticed an increasing apathy and generally felt off-colour. She consulted her general practitioner, who put her to bed for 6 days. At the time she was pyrexial (101° F). Following the bed rest, she noticed increasing stiffness of her back and neck and pain in both her thighs and calves, which were also tender. She then noticed weakness of her right arm. Thinking that these symptoms were due to her stay in bed, she decided to attend a 'gym' class which she had previously attended. After an hour's exercise, her symptoms were very much worse; the weakness became more pronounced and it was this symptom which led to her admission to hospital as a case of poliomyelitis.

Examination. An adult female, in considerable pain. Apyrexial. She had marked neck stiffness and back stiffness. A seventh-nerve palsy was present on the right. The right upper limb was definitely weak, the muscles maximally involved being the triceps and biceps. Her grip on the right was weak as well. Right quadriceps weak. Superficial and deep sensation was unimpaired. Of the reflexes, only her ankle jerks could not be elicited. In her arms both biceps and triceps reflexes were present and equally exaggerated. The weakened muscles were tender to palpation.

The course of her illness was more prolonged than any of the other cases seen in this group. She was afebrile throughout her stay in hospital.

Progress. The seventh-nerve weakness was the first to clear up. This occurred within 4 days of her admission. The weakness of her legs was the next to respond, and this took 10 days. This was followed by return of power in the arms 3 days later. The back stiffness persisted, and was still present at the time of discharge.

Investigations. (1) WBC 12,400 per c. mm. ESR 5 mm. in 1 hour. (2) Lumbar puncture: No cells. 22 mg.% of total protein. (3) Stools: No poliomyelitis or Coxsackie virus.

DISCUSSION

Most of these patients resembled cases of poliomyelitis. In all cases, however, prodromal symptoms were inconspicuous and fleeting. Fever was slight, and muscle weakness was seldom profound. Muscle pains were severe. Absent or depressed reflexes were common. We considered Coxsackie-virus infections but excluded this diagnosis on virology. Guillane Barre's disease or infectious polyneuritis remained an obvious diagnosis. Against this condition, however, was the severe neck and back stiffness not commonly encountered in the former condition. The invariably normal cerebrospinal fluid also excluded Guillane Barre's disease.

In no single case was there a previous history of overt poliomyelitis with paralyses. In no case could we establish poliomyelitis contacts. The possibility remained that the condition might be modified poliomyelitis, or perhaps an allergic response to the poliomyelitis virus, which was circulating in Johannesburg at that time. In no single case was the poliomyelitis virus found in the stool, so that the allergic response might have been merely from some form of contact.

Illnesses comparable to our series are described in the literature. Macrae and Galpine (1954) described 13 similar cases in nurses who had been nursing cases of poliomyelitis. The symptoms were similar to those of the cases described above. Acheson (1954) described another outbreak in a

nurses' home amongst nurses not actively nursing poliomyelitis. Sigurdsson *et al.* (1950), Pellow (1951), White and Burtch (1954), all describe similar outbreaks. Sigurdsson *et al.* describe an epidemic incidence in Iceland of 6·7 per 100 of population in the area involved. In all these outbreaks the common features were as follows:

All cases occurred in a poliomyelitis epidemic. The disease was mild, with no mortality and no permanent paralyses. Muscle pain and tenderness were severe. Pyrexia was slight, or absent, and the cerebrospinal fluid was normal or showed only slight changes.

It would appear that in association with poliomyelitis there circulates a similar, but modified, illness. This may be a form of poliomyelitis which has undergone changed characteristics; or, more likely, it may be an allergic illness, the allergic reagent being the poliomyelitis virus.

SUMMARY

Fourteen cases of a poliomyelitis-like condition are described. The characteristic features were:

1. Headache; pain and stiffness of the neck and back; weakness and tenderness of muscles; slight pyrexia or none at all; and a normal cerebrospinal fluid.
2. The condition occurred in the course of an epidemic of poliomyelitis in Johannesburg.
3. No permanent sequelae were noted and no mortality.
4. No poliomyelitis or other virulent pathogen was found.
5. The resemblance between this outbreak and others elsewhere in the world is noted.

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GESIGSPYN VAN DIE MIGRAINE-TIPE

BESKRYWING VAN 'N GEVAL

R. LIPSCHITZ, F.R.C.S. en D. HAGEN, M.B., CH.B.

Departement van Chirurgiese Neurologie, Baragwanath-Hospitaal

Tipiese migraine is seldsaam in die Bantoe beskryf en gesigspyn van die migraine-tipe waarskynlik nog meer seldsaam. Verslag oor so 'n geval word hiermee aangebied.

Op 12 Februarie 1957 is 'n Bantoe-man (Sotho) van 48 jaar in die Departement van Chirurgiese Neurologie, Baragwanath-Hospitaal opgeneem. Tot Desember was die pasiënt volkome gesond toe hy aangemeld het met die klagte van hoofpyn, soortgelyk aan sy huidige klagte. Toentertyd het die pyn vir 2 maande geduur waarna dit vanself verdwyn het. 'n Serebrale angiogram was gedoen en die pasiënt beweer dat daar aan hom gesê was dat dié ondersoek negatief was—die pasiënt is besonder intelligent.

In Desember 1956 het hy weer aanvalle van ‚migraine' begin kry. Die aanvalle het om 3-uur vm. begin wanneer dit hom wakker maak. Soms was dit voorafgegaan deur 'n gevoel van spelde en naalde in sy linker-arm en -been met flikkerligte in sy linkerhandse gesigsveld. Kort daarna kry hy 'n skielike kloppende pyn oor sy regtervoorkop, boonste deel van regterkant van neus en regtertempel. Die pyn het nooit na die linkerkant versprei nie. Die hoofpyn het geleidelik verskyn en opgewerk tot maksimale intensiteit, het van een tot verskeie ure geduur en het dan ewe skielik verdwyn. Die tydperk tussen die aanvalle was geheel en al pyn-vry. Vanaf Desember 1956 totdat hy onder ons behandeling gekom het, 2 maande later, het die aanvalle elke nag klokslag hulle opwagting gemaak. Gedurende die aanval water sy regteroog, die regterkant van die neus swel toe, sy wang voel warm en die tempel-are aan dieselfde kant staan sigbaar uit.

Die enigste ander punte wat op die geskiedenis betrekking het, was, dat hoewel die pasiënt regshandig is, sy een broer links is; daar is egter geen familiegeskiedenis van epilepsie nie.

By Ondersoek: Tussen die aanvalle was daar geen neurologiese abnormaliteit te bespeur nie. Gedurende 'n aanval sou sy regteroog water en die regterwang het warmer gevoel. Hart, longe en buik was klinies negatief. Bloeddruk 110/70 mm. Hg. Pols 84 per minuut. Asemhaling 20 per minuut.

Spesiale ondersoeke: Röntgenfotos van skedel en borskas was negatief.

In ooreenstemming met bogenoemde kliniese bevindings, het ons besluit om die pasiënt met onderhuidse inspuiting van dehydroergotamine tartraat te behandel. Om 2-uur voormiddag vir 5 agtereenvolgende dae het hy 'n inspuiting van die preparaat ontvang. Aanvangsdosis was 0·25 mg. (in 1 k.s.) opgewerk na 0·5 mg. (in 2 k.s.). Gedurende hierdie tydperk was hy heeltemal vry van pyn en na 5 dae is die behandeling gestaak. Met die skryf van hierdie artikel, 'n maand later, was hy nog pyn-vry.

BESPREKING

Hierdie besondere tipe hoofpyn is deur verskeie outoriteite onder verskillende benamings beskryf. So byvoorbeeld kan ons meld Harris,[1] Symonds,[2] Dott[3, 4] en Horton.[5] Die kondisie was genoem, ‚periodic migrainous neuralgia', ‚ciliary (migrainous) neuralgia', ‚recurrent brief headache of „cluster" pattern', ‚a particular variety of headache', ‚facial migrainous neuralgia'—laasgenoemde is die terminologie wat in hierdie artikel gebruik is.

Meeste outoriteite neem aan dat die toestand primêr die takke van die arteria carotus externa aantas en 'n migrainemeganisme ten gronde het.

Aura, soos in die vermelde geval, is nie kenmerkend van die toestand nie. Gesigspyn van die migraine-tipe kan egter gepaard gaan met 'n aantassing van die inwendige serebrale vate, in welke geval daar wel aura intree.[2]

Differensiële Diagnose

Onder andere moet die volgende kondisies aandag geniet:

1. Siekte en trauma van die oog, oor, neus en keel en dentisie moet natuurlik uitgesluit word.

2. Kwaadaardige patologie wat die takke van die nervus trigeminus aantas, beide binne en buite die skedelholte.

3. *Tic douloureux*—hierdie kwaal tas primêr die vyfde kraniaalsenuwee aan. Dié pyn is paroksismaal, geweldig skielik in aanvang en van 'n warm, skerp en stekende geaardheid. Daarby is die pyn besonder periodiek met kenmerkende ‚afspring areas' (trigger zones). Die pasiënte is gewoonlik ook ouer en die warmtegevoel van die gesig met toe-neus en waterige oog, is afwesig.

4. Temporale arteritis tas die arteria temporalis superficialis aan en vorm 'n deel van wyd verspreide bloedvatsiekte. Procaine-infiltrasie of segmentale reseksie van die aar bring soms verligting.

5. Atipiese gesigspyn sluit verskeie toestande in. 'n Voorbeeld hiervan is chroniese gesigspyn van sentrale oorsprong. In hierdie geval word die hersingstam deur vaskulêre of neuro-degeneratiewe patologie of deur virusinfeksie aangetas. Die pyn is van aanhoudende aard en oorheers soms die hele miserabele bestaan van die pasiënt. Die behandeling is besonder moeilik en gaan soms met *tic douloureux* gepaard.

SUMMARY

1. A case of facial migrainous neuraliga in a Bantu male is reported.

2. A brief discussion on the pathogenesis and differential diagnosis is given.

3. The condition responds typically to ergotamine treatment.

Graag spreek ons ons dank uit teenoor dr. G. D. Elliot, die Agerende Superintendent van Baragwanath-Hospitaal vir sy toestemming om die geval te publiseer en dr. R. F. Flemming, chirurg te Baragwanath-Hospitaal, wie die geval na ons refereer het.

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41STE SUID-AFRIKAANSE MEDIESE KONGRES, DURBAN

KONGRES-NUUSBRIEF

Hierdie brief dien om u daaraan te herinner dat die volgende Wetenskaplike Vergadering en Kongres van die Mediese Vereniging van Suid-Afrika van 16-21 September 1957 in Durban gehou sal word, *wat ongeveer binne 4 maande is*.

By die Openingsplegtigheid van die Kollege van Interniste en Chirurge van Suid-Afrika, wat gedurende verlede Augustus in Johannesburg gehou is, het Sy Edele, die Minister van Gesondheid, in sy toespraak gesê dat die stigting van hierdie na-graadse Kollege 'n bewys is dat, sover dit Interne Geneeskunde betref, Suid-Afrika opgegroei het. Hierdie opmerking word ten volle bekragtig deur die reëlings wat nou getref word vir die komende Kongres wat gedurende September in Durban gehou sal word.

Om verskeie redes sal hierdie Kongres uitstaande wees. Een van die belangriker glansnommers daarvan sal die Wetenskaplike Uitstalling wees. Hierdie Uitstalling sal 'n onafgebroke wetenskaplike en navorsingrolprentvertoning oor 'n groot verskeidenheid van mediese onderwerpe insluit, en onlangse aanbiedinge sluit 'n 16-mm. kleur- en klankfilm, getitel ‚Stress and the Adaptation Syndrome' in. Hierdie rolprent is onder die persoonlike toesig van prof. Hans Selye van Montreal gemaak, en is 'n dokumentêre opsomming van sy uitgebreide navorsings.

Die omvattende Wetenskaplike Program van hierdie Kongres geniet baie belang oorsee en 'n aantal wêreldgesaghebbendes het die uitnodiging van die Plaaslike Organiserende Komitee aangeneem om teenwoordig te wees en aktief deel te neem aan die Wetenskaplike Verrigtinge.

GESIENE GASTE

Die volgende besoekers het hulle voorneme uitgespreek om die Kongres in September by te woon:

Sir Russel Brain, President van die Royal College of Physicians of London.

Sir Harry Platt, President van die Royal College of Surgeons of England.

Prof. E. L. Bortz, Departement van Interne Geneeskunde, Universiteit van Pennsylvania, V.S.A.

Prof. Alexander Brunschwig, Cornell-Universiteitskollege van Interne Geneeskunde, New York.

Dr. R. Bryce-Smith Nuffield-Departement van Anestese, Oxford, Engeland.

Dr. H. Churchill-Davidson, Anestese-Departement, St. Thomas-Hospitaal, Londen.

Dr. J. H. Cyriax, Internis by die Departement van Fisiese Geneeskunde, St. Thomas-Hospitaal, Londen.

Dr. D. M. T. Gairdner, Konsulterende Kinderarts, Addenbrook-Hospitaal, Cambridge, Engeland.

Dr. Michael Gelfand, O.B.E., Chirurg, Algemene Hospitaal, Salisbury, S.-Rhodesië.

Dr. William Gissane, Chirurg by die Birmingham-Ongevalle-Hospitaal, Engeland.

Prof. T. Pomfret Kilner, C.B.E., Nuffield-Departement van Plastiese Chirurgie, Oxford, Engeland.

Prof. V. Kinross-Wright, Departement van Psigiatrie, Baylor-Universiteit, Houston, Texas.

Dr. Charles Marks, Chirurg, Algemene Hospitaal, Salisbury, S.-Rhodesië.

Prof. Alan Moncrieff, C.B.E., Direkteur van die Instituut vir Kindergesondheid, Universiteit van Londen.

Dr. A. Killey Monro, Lektor in Chirurgie by die Na-graadse Hospitaal, Hammersmith, Londen.

Dr. J. S. Richardson, M.V.O., Internis, St. Thomas-Hospitaal, Londen.

Prof. Paul Wilflingseder, Departement van Chirurgie, Universiteit van Innsbrück, Oostenryk.

Dr. A. J. Wrigley, Verloskundige Chirurg, St. Thomas-Hospitaal, Londen.

VOLTALLIGE SITTINGS

Die onderwerpe wat vir die Voltallige Sittings gekies is, is van die grootste belang vir almal teenswoordig. Die onderwerpe en sprekers sal as volg wees:

1. ‚*Cerebral Vascular Disease and the Problem of Aging*'.
   (*a*) Sir Russel Brain, President van die Royal College of Physicians of London.
   (*b*) Dr. M. M. Suzman, Johannesburg.
   (*c*) Dr. F. H. Kooy, Kaapstad.
   (*d*) Dr. Keith L. Allen, Johannesburg.
   (*e*) Prof. E. L. Bortz, Philadelphia.
2. ‚*The Parasitic Diseases of man in Africa*'.
   (*a*) Dr. Michael Gelfand, O.B.E., Salisbury, S.-Rhodesië.
   (*b*) Dr. Charles Marks Salisbury, S.-Rhodesië.
   (*c*) Dr. R. Elsdon-Dew, Durban.
3. ‚*The Surgery of Repair*'.
   (*a*) Sir Harry Platt, President van die Royal College of Surgeons of England.
   (*b*) Dr. William Gissane, Birmingham, Engeland.
   (*c*) Prof. T. Pomfret Kilner, C.B.E., Oxford, Engeland.

4. ‚*Recent Advances in Child Care*'.
   (*a*) Prof. Alan Moncreiff, C.B.E., Londen.
   (*b*) Dr. D. M. T. Gairdner, Cambridge, Engeland.
   (*c*) Dr. H. L. Wallace, Durban.

WETENSKAPLIKE AFDELINGSVERGADERINGS

Afgesien van hierdie Voltallige Sittings, sal daar talryke Wetenskaplike Afdelingsvergaderings oor al die erkende spesialiteite, beide van Interne Geneeskunde en Chirurgie, wees, by welke geleenthede gevorderde en oorspronklike werk vir bespreking en hersiening aangebied sal word.

Dit is nog te vroeg om enige van die uitstekende referate wat tot dusver aangebied is, vir kommentaar uit te kies, maar dit word verwag dat ‚*New Horizons in Chemotherapy*' deur prof. V. Kinross-Wright van die Departement van Psigiatrie, Baylor-Universiteit, Houston, Texas, V.S.A. meer as net betyds sal wees. ‚*Recent Advances in Aviation Medicine*' en ‚*Medical Aspects of Atomic Warfare*', wat deur die Senior Uitvoerende Raad en Gesondheidsbeamptes van die Unie-verdedigingsmag aangebied sal word, tesame met kleurrolprente wat tot nog toe nie vrygestel is nie, is gekombineerde Afdelingsvergaderings wat sekerlik baie aandag sal geniet.

DIE LIGTER SY VAN KONGRES

Die Dames-Hulpkomitee, onder die bekwame Voorsitsterskap van mev. H. Grant Whyte, doen alles in hulle vermoë om 'n afwisselende sosiale program te verskaf en om te verseker dat almal in staat sal wees om te ontspan en hulle verblyf in Durban te geniet.

Sosiale Plegtighede

Maandag, 16 September, 8.30 nm. Openingsplegtigheid (aanddrag en akademiese drag).
Dinsdag, 17 September, 8.30 nm. Kongresfeesmaaltyd.
Woensdag, 18 September, 6.30 nm. Burgerlike Ontvangs.
Donderdag, 19 September, agtermiddag, Campbell-Watt-Trofee (Gholf), en Casewell-Struthers-Trofee (Rolbal).
Vrydag, 20 September, 8.30 nm. Kongresdansparty (aanddrag).

Onthaal

Die Voorsitter van die Clairwood-Renbaan het welwillend aangebied om ere-lidmaatskap van die Renbaan aan lede van die Vereniging toe te staan vir die perdewedrenne op Saterdag, 14 September en Saterdag, 21 September. Die gesiene besoekers sal die gaste van die baanbeamptes wees.

Die Durban-Klub het ere-lidmaatskap, en die fasiliteite van die Dameseetsalon aan die here van die Vereniging verleen.

Die Joodse Klub het ere-lidmaatskap aan lede van die Vereniging en hulle vrouens aangebied. Fasiliteite is ook deur die Durban Country-Klub, die Royal Natal Yacht-Klub en die Kloof Country-Klub aangebied.

Damesprogram

'n Program van vermaaklikheid en uitstappies vir besoekende dames is deur die Dames-Hulpkomitee voorberei en sluit in uitstappies na plekke van spesiale belang, 'n modeparade, lesings oor binnehuise versiering en kookkuns, 'n Simfoniekonsert en 'n Verskeidenheidskonsert. 'n Tennistoernooi en 'n Brugwedstryd is ook gereël.

Die Feesmaaltyd

'n Feesmaaltyd vir Mediese Gegradueerdes sal op Dinsdagaand, 17 September in die Stadsaal, Durban, gehou word by welke geleentheid Sy Eksellensie, die Goewerneur-Generaal, sy voorneme uitgespreek het om aanwesig te wees. Hierdie funksie sal lede van die Vereniging 'n geleentheid gee om ons gesiene oorsese gaste informeel aan te hoor en te ontmoet. Spesiale reëlings sal getref word dat mede-alumni, lede van Groepe en vriende bymekaar kan sit en die kans sal hê om vriendskapsbande te hernieu.

Die Dansparty

Die Kongresdansparty sal op Vrydagaand, 20 September, om 8.30 nm. in die Stadsaal, Durban, gehou word. Spesiale reëlings word getref om te verseker dat hierdie funksie die uitstaande hoogtepunt van die sosiale programme van die 1957-Mediese Kongres sal wees.

See-Uitstappies

Vir diegene met meer ondernemingsgees, het die Hawebeamptes aangebied om elke aand, gedurende die Kongres, seevarende sleepbote beskikbaar te stel sodat lede van die Vereniging 'n geleentheid sal hê om hulle ‚see-tyd' te doen.

Spesiale Datumseël

Die verskillende Staats- en plaaslike beamptes het alles in hulle vermoë gedoen om seker te maak dat 'n besoek aan Durban gedurende Kongresweek 'n uiters genotvolle ondervinding en 'n blywende herinnering sal wees. Dit mag van belang vir posseël-versamelaars wees dat die Posmeester-Generaal 'n spesiale datumseël van 16 tot 21 September 1957, vir die gebruik van die Mediese Kongres, goedgekeur het.

BELANGRIK

1. Lede wat van plan is om die Kongres by te woon en nog nie te kenne gegee het dat hulle dit wil doen nie, moet hulle voornemekaartjie dadelik voltooi en terugstuur.

2. Lede wat van plan is om referate voor te lees, word daaraan herinner dat 'n getikte samevatting daarvan, wat nie 500 woorde oorskrei nie, nie later as 18 Junie 1957 aan die ere-mediese sekretarisse voorgelê moet word nie, en 'n getikte afskrif in duplikaat van die voltooide referaat, nie later as 18 Julie 1957 nie.

3. Moontlike besoekers moet, indien hulle dit nog nie gedoen het nie, dadelik huisvesting bespreek. Daar is nog huisvesting in Durban beskikbaar.

Mediese Sentrum 53,
Fieldstraat,
Durban

Mei 1957.

Ere-Organiserende Sekretarisse
Dr. B. Crowhurst Archer
Dr. S. Disler

Ere-Mediese Sekretarisse
Dr. J. Kelman Drummond
Dr. A. J. Wilmot

Telegramme ‚Medcongres'
Telefoon 6-8415
Na-ure 5-6131

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

AMENDMENT OF BY-LAW 58

At the meeting of the Federal Council held in Johannesburg on 29 March 1957, Council agreed that By-Law 58 be amended to read as follows:

'There shall be constituted a Head Office and Journal Committee which shall consist of the Cape Western Branch Members of the Federal Council with powers to coopt.

The Head Office and Journal Committee shall have power to deal with all matters affecting the administration of the *Journal* and Head Office, provided that without the prior consent of the

WYSIGING VAN VERORDENING 58

Op die vergadering van die Federale Raad te Johannesburg op 29 Maart 1957 gehou, het die Raad besluit om Verordening 58 te wysig sodat dit lui as volg:

‚Daar moet 'n Hoofkantoor- en Tydskrifkomitee wees wat moet bestaan uit die Wes-Kaaplandse taklede van die Federale Raad, met die reg om te koöpteer.

Die Hoofkantoor- en Tydskrifkomitee is by magte om met alle sake te handel wat die administrasie van die *Tydskrif* en die Hoofkantoor raak, met dien verstande, egter, dat, sonder die

Federal Council or of the Executive Committee the Head Office and Journal Committee shall not—

(*a*) incur any extraordinary expenditure, or

(*b*) determine or vary the salary scales, remuneration or allowances of any official whose total remuneration exclusive of allowances exceeds £1,000 per annum, or

(*c*) mortgage, sell or purchase any immovable property on behalf of the Association.'

By order of the Council

A. H. Tonkin
Secretary

Medical House
Cape Town
10 May 1957

voorafgaande toestemming van die Federale Raad of van die Uitvoerende Komitee, mag die Hoofkantoor- en Tydskrifkomitee nie—

(*a*) enige buitengewone uitgawe aangaan nie.

(*b*) die salarisskale, besoldiging of toelaes van enige amptenaar wie se totale besoldiging, met uitsondering van toelaes, meer as £1,000 per jaar is, vasstel of verander nie, of

(*c*) enige vaste eiendom namens die Vereniging verhipotekeer, verkoop of koop nie.'

Op las van die Raad

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
10 Mei 1957

IN MEMORIAM

IAN GLOAG, M.B., CH.B.

Dr. S. Disler, Medical Superintendent, King Edward VIII Hospital, Durban, writes: Dr. Ian Gloag was 36 years of age when he passed away at Addington Hospital on 16 March 1957. Before the war he was an engineering student at Cape Town University for a short time. He proceeded on active service from 1940-44 and after demobilization became a medical student at Cape Town, where he qualified in 1953.

He held hospital appointments at the Somerset Hospital and Woodstock Hospital, Cape Town, and then went into private practice. He later moved to Durban and on 5 November 1956 was appointed to the staff of King Edward VIII Hospital, as a Registrar in Anaesthetics. There he was known as a conscientious and hard worker, very keen and energetic, and popular among the staff.

Dr. Gloag was a keen photographer and recently became very interested in tape recording, sending messages to friends in many parts of the world.

He leaves three children of school age. We, at King Edward VIII Hospital extend our deepest sympathy to his family.

PASSING EVENTS : IN DIE VERBYGAAN

Cape Western Branch. A dance will be held at the King David Country Club (near D. F. Malan Airport) on Saturday, 15 June 1957. Tickets are available from the Branch Office, 35 Wale Street, Cape Town (telephone 3-1574).

* * *

Research Forum, University of Cape Town. The next meeting of the Research Forum will be held on Wednesday, 5 June at 12 noon in the A-Floor Lecture Theatre, Groote Schuur Hospital, Cape Town. The speaker will be Dr. H. Gordon (for the Clinical Nutrition Research Unit, University of Cape Town) and the subject, *Further Studies on Dietary Fats and the Serum Cholestrol.*

* * *

University of the Witwatersrand, Johannesburg. Medical Graduates Association. A postgraduate course in Clinical Paediatrics with a special emphasis on case demonstrations will be held on Friday, Saturday and Sunday, 7, 8 and 9 June, under the auspices of the Department of Paediatrics of the University of the Witwatersrand.

For details apply to the Secretary, Medical Graduates Association, Medical School, Johannesburg, telephone 44-7040 (mornings).

* * *

South African Paediatric Association. The next meeting of the Cape Town Sub-group of the Association will take the form of a Clinical Evening to be held at the Princess Alice Home of Recovery, Retreat, Cape, on Tuesday, 4 June 1957, at 8.15 p.m. All practitioners are welcome.

* * *

Mr. Cecil Morris, Orthopaedic Surgeon, Johannesburg, has left for Lambarene, French Equatorial Africa, where he will be spending a few weeks at the Hospital of Dr. Albert Schweitzer. During his absence Mr. G. Fischer will act as his locum.

* * *

The Faculty of Radiologists of the South African College of Physicians and Surgeons. At the request of the College of Physicians and Surgeons a meeting of Radiologists who are foundation fellows or members of the College of Physicians and Surgeons was called to organize the Faculty of Radiologists. The following were elected as the organizing committee:

Chairman, Dr. M. Weinbren. *Secretary-Treasurer*, Dr. P. J. Loots. *Members of the Committee*, Dr. Eric Samuel, Dr. M. H. Fainsinger and Dr. M. Shapiro for the Transvaal, Dr. J. M. Grieve for the Cape, Dr. J. Coller for Natal and Dr. F. Gillwald for the Orange Free State. A number of meetings have been held and a draft constitution is to be submitted to the Council of the College of Physicians and Surgeons shortly. Any information required concerning the Faculty of Radiologists, should be addressed to Dr. P. J. Loots, P.O. Box 1756, Johannesburg.

* * *

Dr. J. J. de Waal, F.R.C.P.E., D.P.H., D.T.M., praktiseer nou as 'n internis te Harley Chambers, 207 Jeppestraat, Johannesburg. Telefone, Spreekkamers 22-1666, Woning 42-4810.

* * *

Dr. M. M. Suzman, M.D., of Johannesburg, has been elected to the Fellowship of the Royal College of Physicians of London.

* * *

Dr. Isaac Cohen, M.B., B.Ch. (Rand), M.R.C.P. (Edin.), has commenced practice as a specialist physician at 68b Lister Buildings, Jeppe Street, Johannesburg. Telephone 22-0950.

* * *

Unie van Suid-Afrika. Aangifte van ernstige epidemiese siektes en poliomiliëtis in die Unie gedurende die tydperk 3 Mei-9 Mei 1957.

Poliomiëlitis

	Bl.	*Nat.*	*Kl.*	*As.*	*Totaal*
Transvaal	10	6	1	1	18
Kaapprovinsie ..	10	7	17	—	34
Oranje-Vrystaat ..	2	4	—	—	6
Natal	6	7	—	1	14
Totaal ..	28	24	18	2	72

Pes, Pokkies, Tifuskoors: Geen.

REVIEWS OF BOOKS : BOEKRESENSIES

BACTERIOLOGY, MYCOLOGY AND PARASITOLOGY

Medical Bacteriology including Elementary Mycology and Parasitology. Sixth Edition. Sir Lionel Whitby, C.V.O., M.A., M.D. (Camb.), F.R.C.P. (Lond.), D.P.H. and Martin Hynes M.D. (Camb.), M.R.C.P. (Lond.). Pp. viii + 540. 103 Illustrations. 30s. London: J. & A. Churchill Ltd. 1956.

Contents: 1. The General Properties of Bacteria. 2. Sterilization. 3. The Cultivation of Bacteria. 4. Methods of Examining Bacteria. 5. Infection and Resistance. 6. The Basis of Immunity. 7. Hypersensitiveness. 8. Practical Applications of Immunology. 9. Chemotherapy and Antibiotics. 10. Common Contaminating Organisms. 11. The Cocci. 12. Intestinal Bacteria. 13. Corynebacterium. 14. Mycobacteria. 15. Actinomyces and Related Bacteria. 16. Haemophilus. 17. Brucella. 18. Pasteurella. 19. Aerobic Spore-Bearers. 20. Anaerobic Spore-Bearers. 21. The Spirochaetes. 22. Miscellaneous Organisms. 23. Rickettsia. 24. The Viruses. 25. Diseases Produced by Fungi. 26. Protozoal Diseases. 27. Helminth Infections. 28. Arthropod Vectors of Disease. 29. The Collection and Examination of Specimens. 30. The Bacteriology of Water, Milk and Food. Appendix. Index.

Those who have made use of this book in the past will not be surprised to hear that the authors have prepared a new edition and that the chapters on the antibiotics and fungi have been rewritten; both chapters are greatly improved. They will notice however that in the publisher's slip on the paper dust-cover it is claimed that there is also a 'new section added on the arthropod vectors'. The form and the remaining chapters are almost exactly as before.

For those who have not encountered this book or one of its predecessors, it must be pointed out that the subjects of Medical Bacteriology including elementary Mycology and Parasitology are dealt with in 526 pages in such a way that the information is presented in synoptic fashion. Too little emphasis is placed on the pathogenesis and epidemiology of the various infectious diseases as is illustrated by the 14 lines devoted to the former and 20 lines devoted to the latter in the section dealing with poliomyelitis.

The subject is 'presented as a part of medicine including the view point of the clinical pathologist'. This may well have been the intention of the authors but in the absence of a bibliography there is no reference to other sources, recent or remote, where the reader might gather a little more information than that contained in this terse account.

The medical students' curriculum is a very full one these days so there will be some who will find this book contains enough to satisfy their examiners, but for others who might wish to use it, its brevity will prove a shortcoming.

A.K.

OPERATIVE SURGERY ILLUSTRATED

Operative Surgery. Volume I. Under the General Editorship of Charles Rob, M.C., M. Chir., F.R.C.S. and Rodney Smith, M.S., F.R.C.S. Pp. xviii + 98 (Part I) + 109 (Part II) + 135 (Part III) + (5) (Index). 570 Illustrations. (This work will consist of 8 Volumes at £5 10s. 0d. for each volume and an Index at £2 0s. 0d.) London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1956.

Contents: Introduction by Charles Rob and Rodney Smith. *Part I: Introductory. Section I: General Technique.* Ligature and Suture Materials. Local Anaesthesia—Technique. Posture. Haemostasis. Infusions and Transfusions. Diathermy. Needle Biopsy. Use of Retractors. Methods of Dissection. Drainage. Preparation of the Skin. Technique of Suture. Radium Needle Insertion. Burst Abdomen—Wound Dehiscence. *Part II: Surgery of Trauma. Section I: General Treatment of Wounds.* Excision of Soft Tissue Wounds. Primary, Delayed Primary and Secondary Suture. Primary Suture of Severed Vessel, Nerve and Tendon. Local Care of Burns. *Section II: Bones and Joints.* Open Fractures. Open Injuries to Joints. Use of Skeletal Traction in Fractures. *Section III: Abdomen.* Abdominal Injuries. *Section IV: Genito-urinary Tract.* Injuries to the Kidney. Injuries to the Ureter. Injuries to the Bladder. Injuries to the Urethra. *Section V: Chest.* Penetrating Wounds of the Chest. Surgery of Retained Missiles in the Lung. Injuries of the Pericardium and Heart. Injuries to the Oesophagus. *Section VI: Head Wounds.* Wounds of the Face and Jaws. *Part III: Abdomen. Section I: Introductory.* Surgery of Access. Exploration of Abdomen: Acute. Exploration of Abdomen: Non-Acute. Specimen Anastomosis of Hollow Viscera. *Section II: Stomach and Duodenum.* Gastroscopy. Pyloromyotomy in Infants. Gastrostomy. Simple Closure of Perforated Peptic Ulcer. Gastro-Duodenostomy. Pyloroplasty and Pyloromyotomy. Duodeno-jejunostomy. Gastro-jejunostomy and Antral-exclusion. Partial Gastrectomy. Operations for Bleeding Peptic Ulcer. Gastro-jejunal Ulcer and Gastro-jejuno-colic Fistula. Gastrectomy for Cancer. Vagotomy. Index.

In the first volume of this new comprehensive work, the authors have attempted to teach operative surgery by the visual method. Each operation is described in pictures stage by stage; the accompanying text is very brief and reliance is placed on the various illustrations to explain the operations.

In assessing the value of this work, the authors' introductory remarks must constantly be kept in mind. They state: 'By far the best way of learning to perform an operation is to watch, or better still, to assist an acknowledged master carry out the procedure on several occasions. Nothing contained in a book can equal a personal apprenticeship of this kind.' Furthermore, it is clearly stated that the work should be complementary to *British Surgical Practice* in which the indications, pre-operative and post-operative treatment, complications, etc. of the various operations are dealt with in detail.

If these two reservations are accepted, the work is to be welcomed as a useful addition to the literature on operative surgery. The illustrations on the whole are good, although some of them tend to be rather diagrammatic. The procedures outlined are those practised by recognized experts and, although one may disapprove of certain techniques on the grounds that they differ from one's own personal methods, they are obviously acceptable and sound.

The chapters in Vol. 1 cover General Techniques, the Surgery of Trauma, and certain aspects of abdominal surgery. All the chapters on general techniques are good and many useful hints are given on suture materials, posture and operative technique. There are some omissions—for instance, the left semi-lateral position for biliary surgery is not mentioned—but on the whole the work is complete. The scanty remarks on haemophilia and Christmas disease need some elaboration and also some revision.

The chapters on the surgery of trauma are excellent and among these Wallace's contribution on burns and that of Lowden on abdominal injuries merit special mention.

Tanner's contributions on gastric surgery are, as always, excellent, and apart from the chapter on duodeno-jejunostomy, the other chapters on abdominal surgery are all good.

This first volume certainly shows good promise and, if the remainder of the work is of a similar standard, it should adequately fulfil its purpose, i.e. to help the surgeon who is called upon to operate in an unfamiliar field and the postgraduate student during his training period as a surgical registrar.

J.H.L.

WILLIAMS' OBSTETRICS

Williams' Obstetrics. Eleventh Edition. By Nicholson J. Eastman. Pp. x + 1212. Illustrations. New York: Appleton-Century-Crofts Inc. 1956.

Contents: Preface. *Section One: Orientation.* 1. Obstetrics in Broad Perspective. *Section Two: Anatomy and Physiology of Re-production.* 2. The Anatomy of the Female Reproductive Organs. 3. The Ovarian Cycle and Its Hormones. 4. The Endometrial Cycle and Menstruation. 5. The Physiology and Development of the Ovum. 6. The Placenta and Its Hormones. 7. The Development and Physiology of the Fetus. 8. Maternal Physiology in Pregnancy. *Section Three: Management of Normal Pregnancy.* 9. Diagnosis of Pregnancy. 10. The Normal Pelvis. 11. Lie, Presentation and Position of the Fetus: Methods of Diagnosis. 12. Antepartal Care. 13. Psychiatric Aspects of Pregnancy and Childbirth. *Section Four: Physiology and Conduct of Labor.* 14. The Forces Concerned in Labor. 15. The Mechanism of Labor in Vertex Presentations. 16. The Clinical Course of Labor. 17. The Conduct of Normal Labor. 18. Analgesia and Anesthesia. *Section Five: The Puerperium and the Newborn.* 19. The Puerperium. 20. The Newborn. *Section Six: Abnormalities of Pregnancy.* 21. Abortion and Premature Labor. 22. Ectopic Pregnancy. 23. Diseases and Abnormalities of the Fetal Membranes and Placenta. 24. Placenta Previa; Abruptio Placentae; Obstetric Shock. 25. Complications Due to Diseases and Abnormalities of the Generative Tract. 26. Multiple Pregnancy. 27. Nausea and Vomiting of Pregnancy. 28. The Toxemias of Pregnancy. 29. Coincidental Complications of Pregnancy. *Section Seven: Abnormalities of Labor.* 30. Dystocia Due to Anomalies of the Expulsive Forces. 31. Dystocia Due to Abnormalities in Position, Presentation or Development of the Fetus. 32. Dystocia Due to Pelvic Contraction. 33. Dystocia Due to Abnormalities of the Generative Tract. 34. Injuries of the Birth Canal. 35. Abnormalities of the Third Stage of Labor. *Section Eight: Abnormalities of the Puerperium.* 36. Puerperal Infection. 37. Disorders of the Puerperium Other Than Puerperal Infection. *Section Nine: Abnormalities of the Newborn.* 38. Iniuries Sustained by the Infant in Pregnancy and Labor. 39. Diseases and Malformations of the Newborn. *Section Ten: Operative Obstetrics.* 40. Artificial Termination of Pregnancy and Sterilization. 41. Forceps. 42. Breech Extraction and Version. 43. Cesarean Section. 44. Destructive Operations. Index.

This textbook of obstetrics, surely one of the finest on the market, comes from the pen of the professor of obstetrics at the Johns Hopkins University. It is a beautiful book to handle and a pleasure to hold.

The author has succeeded admirably in the main purpose of this new edition, viz., to incorporate the many advances of the 6

years since the previous edition appearing in 1950. There is a new chapter on the psychiatric aspects of pregnancy and childbirth. Among the sections which have been re-written are those on obstetric shock, afibrinogenaemia, the treatment of accidental haemorrhage, habitual abortion, uterine inertia, anaemias, rupture of the Caesarean-section scar, postmaturity, hypotensive drugs, normorphine, uterine blood-flow, retrolental fibroplasia, placental histology, hydatidiform mole and choriocarcinoma, and much of the sections dealing with embryology and endocrinology.

With regard to the now much-discussed problem of prolonged pregnancy, Professor Eastman favours the lapse of 3 weeks (rather than 2 weeks) beyond the calculated date of delivery as the indication of postmaturity. The idea that postmaturity results in infants of excessive size with consequent mechanical dystocia is regarded as unfounded by most clinicians. Even though postmaturity may possibly impose a slightly increased risk of foetal death it is probable that this hazard is less than that associated with the induction of labour in the presence of the unripe cervix. The author therefore feels that postmaturity is not an indication for induction of labour unless the state of the cervix is very favourable, except in the case of the elderly primigravida, who should not be allowed to go more than one week over term, when Caesarean section and not induction of labour is the treatment of choice. One welcomes this sane and balanced viewpoint.

In the treatment of accidental haemorrhage Professor Eastman gives prominent place to early artificial rupture of the membranes. The management of afibrinogenaemia receives due consideration.

There is not much to criticize in this book but one may perhaps mention the description and illustrations of the use of the Voorhees bag in the treatment of placenta praevia, which might well have been omitted.

This is a comprehensive text-book which one would like to recommend primarily to the obstetric specialist and the specialist in training. I know of none better.

E.M.S.

RADIOLOGY OF THE ALIMENTARY TRACT IN INFANTS

Radiology of the Alimentary Tract in Infancy. By Roy Astley, M.B., M.R.C.S., D.M.R. Pp. viii + 287. With Illustrations. 50s. net. London: Edward Arnold (Publishers) Ltd. 1956.

Contents: Part 1. The Oesophagus. I. Oesophageal Atresia. II. Disorders of Swallowing. III. Vascular Compression of the Oesophagus. IV. Mediastinal Cysts: Duplications of the Foregut. V. Oesophagitis: Oesophageal Strictures: Temporary Oesophageal Obstructions. VI. Various Oesophageal Abnormalities. *Part 2. The Diaphragm.* I. Gastro-oesophageal Incompetence. II. Pleuroperitoneal (Postero-lateral) Diaphragmatic Hernia. III. The Large Hiatus Hernia. IV. Various Diaphragmatic Abnormalities. *Part 3. The Stomach.* I. Hypertrophic Pyloric Stenosis. II. Infantile Pylorospasm. III. Post-operative Vomiting in Pyloric Stenosis. IV. Aerophagy: Gastric Elevation: Duodenal 'Slow-down'. V. Gastritis and Duodenitis. VI. Gastro-duodenal Ulceration. VII. Duplications of the Stomach. VIII. Gastric Diverticula. IX. Rare Obstructive Lesions. X. Various Gastric Abnormalities. *Part 4. The Duodenum.* I. Duodenal Obstructions. II. Annular Pancreas. III. Various Duodenal Abnormalities. *Part 5. The Small Intestine and Peritoneum.* I. Small Intestinal Obstruction. II. Peritonitis and Perforation of the Gut. III. Gastro-enteritis. IV. Meckel's Diverticulum and Related Conditions. V. Various Small Intestinal Abnormalities. *Part 6. The Colon.* I. Congenital Obstructions of the Colon. II. Imperforate Anus and Related Conditions. III. Intussusception. IV. Megacolon. V. 'Meconium Blockage'. VI. Various Abnormalities of the Colon. *Part 7. Miscellaneous.* I. The Liver and Biliary Tract. II. The Spleen, Pancreas, Mesentery and Omentum. III. Retroperitoneal and Pelvic Structures. Index.

Dr. Astley has written a most readable and informative book on the radiological findings in the abnormalities of the alimentary tract in infancy. All the developmental and acquired lesions of the tract in infants have been adequately surveyed and the majority illustrated by excellent case histories.

The author has described in the fullest detail the methods of radiological investigation and the subsequent assessment of the X-ray findings. Many useful hints are given and customary techniques have been elaborated to cope with the frequent difficulties in the examination of infants. Dr. Astley has refuted the contention that the use of barium in examination beyond the oesophagus is hazardous. In his experience, with the use of minimal amounts of barium and subsequent stomach washouts, there has been no significant aspiration into the lungs in several thousands vomiting babies. This book is profusely illustrated with excellent radiographic reproductions. The references appended to each chapter are extensive.

The publication is one which can be highly recommended not only to the radiologist but to the paediatrician and the paediatric surgeon.

J.N.J.

TUBERCULOSIS

Tuberculosis in the Commonwealth 1955. Transactions of the Fourth Commonwealth Health and Tuberculosis Conference, June 1955. Pp. xii + 507. 14 Plates. 30s. London: National Association for the Prevention of Tuberculosis, 1956.

Contents: Foreword. NAPT Council and officers. Photographs. The Preventive Outlook today. Child Hygiene and infection. Some aspects of defferential diagnosis of Pulmonary Tuberculosis. Choice of Drugs in medical and surgical Treatment of Tuberculosis. Do Death Rates Matter? X-ray and Tuberculin Surveys— their meaning and interpretation. Tuberculosis in industry. Tuberculosis—A Problem of Different Races. The Psychology of the Patient and relatives as a factor in successful Treatment. Tuberculosis and Leprosy. Meetings on mass Radiography. Meeting for Members of care Committees and all interested in Social and Voluntary work. Meeting for social workers and health Visitors. Discussion for Sanatorium Matrons and Nurse Teachers. Publicity. Social Arrangements. Special Hospitality. Tours and Visits. Cinema Films. List of Exhibitors. List of Members of conference.

This soft-cover volume, is a full report of the Fourth Commonwealth Health and Tuberculosis Conference, held in the Royal Festival Hall, London, in mid-1955. Organized by the National Association for the Prevention of Tuberculosis, this was a most ambitious effort. The conference was attended by 1,700 delegates representing some 60 countries—35 within the Commonwealth.

The enormous field covered in the sectional discussion is obvious from the list of contents; so much so, that nobody, medical or lay, who is even remotely interested in or associated with tuberculosis will fail to find something new and interesting in it. A prodigious amount of valuable information is contained in the 500 pages of this book. This was achieved by limiting addresses to 10 minutes, so that many more experts could participate in the discussions. Time did not allow an exhaustive or deep consideration of each subject, but this has the distinct advantage of yielding an eminently readable report which keeps the reader's interest throughout.

Perhaps the main value of the book lies in its clear demonstration of the modern trends in tuberculosis control—both preventive and clinical. All these features combine to make it a most valuable reference book, which should find a place among the books of all those who deal with tuberculosis in its many different aspects.

H.R.A.

'ULTRAMICRO' CHEMICAL PATHOLOGY

Ultramicro Methods for Clinical Laboratories. By Edwin M. Knights, Jr., M.D., Roderick, P. MacDonald, Ph.D. and Jaan Ploompuu. Pp. viii + 128. 18 Figures. $4·75. New York: Grune & Stratton, Inc. 1957.

Contents: Foreword. Preface. 1. Setting up Ultramicro Chemistry in the General Laboratory. 2. Special Equipment. 3. Collection of Blood and Pipetting. 4. Sodium and Potassium. 5. Calcium. 6. Chlorides: Serum and Spinal Fluid. 7. Inorganic Phosphate. 8. Carbon Dioxide Combining Power. 9. Blood Urea Nitrogen. 10. Glucose. 11. Total Cholesterol. 12. Acid and Alkaline Phosphate. 13. Bilirubin. 14. Icterus Index. 15. Thymol Turbidity. 16. Cephalin Flocculation Test. 17. Proteins: Total Serum, Serum Albumin and Globulin, and Cerebrospinal Fluid. 18. Electrochromatography. 19. The C-Reactive Protein Test. 20. Microflocculation Tests for Syphilis. 21. Microhematocrits. Appendix: Table of Normal Values. Directory of Distributors and Producers. Index.

In this book the authors describe a system of 'ultramicro' biochemical analysis for clinical laboratories. Ultramicro methods require 10-25 microlitres of blood or serum; a reasonably complete chemical survey can therefore be made on as little as half a millilitre of blood, a quantity easily obtainable as a rule by puncture of the finger tip or the heel. The attraction of such methods is particularly great in work with children and small experimental animals and in all cases when blood is difficult to get and repeated observations are needed for the control of treatment.

The use of such small samples requires the greatest care at all stages. The authors recommend that ultramicro methods should not displace ordinary methods but that a special section of a routine laboratory should be organized for them to be used in difficult cases; this section should have its own space set aside for it, away if possible from the hurly-burly of general routine. It should have a technician whose sole duty it is to be responsible for its maintenance and running. Success will not be achieved with ultramicro methods if they are regarded as something to be dragged out of some hole or corner in an emergency by any technician available.

For these methods special apparatus will be required. Micropipettes, micro-burettes and a micro modification of the Van

Slyke blood-gas apparatus are used. Most of the methods are colorimetirc but the volume of the final solutions are small and require the use of micro-cells in one of the larger and more accurate spectrophotometers.

This book should be very helpful to any pathologist who contemplates establishing such a section in his laboratory and especially to the pathologist to a children's hospital. Individual methods may be of use to experimentalists working with small animals.

G.C.L.

THE MICROSCOPE

Lecture Notes on the Use of the Microscope. 2nd Edition. By R. Barer, M.C., M.A., B.Sc., M.B., B.S. Pp. vii + 76. 8 Figures. 7s. 6d. Oxford: Blackwell Scientific Publications. 1956.

Contents: I. The Microscope Stand and Components. II. How the Image is Formed. III. The Theory of the Microscope. IV. The Lenses of the Microscope. V. Practical Instructions. VI. Practical Instructions (continued). VII. Further Comments on Microscope Illumination. VIII. The Care and Cleaning of the Microscope. IX. How to Look at Living Specimens and Unstained Material. X. Miscellaneous Questions, Hints and Tips. Suggestions for Further Reading. Appendix I.—Some Instructive Experiments. Appendix II.—The Use of Research-type Microscope Lamps.

This little book provides both the fundamental theory of microscopy and practical advice about using a microscope. The theoretical section is a useful revision of the relevant physics, which most medical students and graduates have known at one stage of their careers and subsequently forgotten. The practical section recommends a comprehensive and rather time-consuming routine for setting up and using the microscope; this is useful for the beginner and, with experience, he will develop his own short cuts.

The book will be of value not only to students and technical assistants in the biological sciences but also to their teachers. The style is lucid and even the experienced microscopist will find the approach both novel and stimulating.

A.W.S.

THE LONDON SCHOOL OF TROPICAL MEDICINE

History of the School of Tropical Medicine in London (1899-1949). London School of Hygiene and Tropical Medicine. Memoir No. 11. By Sir Philip Manson-Bahr, C.M.G., D.S.O., M.D., F.R.C.P. Pp. xiv + 328. 30 Illustrations. £2 10s. net. London: H. K. Lewis & Co., Ltd. 1956.

Contents: Preface. *Section I: Historical.* I. Introduction. II. The Scientific Background. III. The Nucleus of the Future School. IV. The Source (The Seamen's Hospital Society). V. Prelude, 1898. VI. Small Beginnings (London School of Tropical Medicine). VII. Progress, 1899-1914. VIII. Set-Back (The First World War 1914-1918). IX. London Venture, 1920-1929. X. Combined Operations, 1930-1939. XI. The Dim Years, 1940-1945 (The Second World War). XII. Resuscitation, 1946-1949. XIII. Functions of the Hospital for Tropical Diseases. XIV. The Story of the School Library. XV. The Story of the School Museum. XVI. The Crucial Malaria Experiment. XVII. Introductory Addresses. XVIII. The Bureau of Hygiene and Tropical Diseases. *Section II: Biographical.* I. Founders of the School. II. Physicians and Lecturers. III. Directors and Superintendents. IV. Deans and Organizers. V. Demonstrators, Pathologists, Research Workers and Bacteriologist. VI. Surgeons and Lecturers. VII. Ophthalmologists. VIII. Dermatologists and Mycologist. IX. Protozoologists. X. Helminthologists. XI. Entomologists. XII. Technicians and Lay Assistants. XIII. Dintinguished Foreign Students at the School. XIV. The Librarian and Curators of the Museum. *Section III: Detailed Diary of the School* 1899-1949. *Section IV: Appendix.* I. Research Expeditions. II. Collected Papers. III. Lists of Prize Winners. IV. The Syllabus. V. Clinical Tropical Medicine (Hospital for Tropical Diseases).

This book is more than a 'History of the School of Tropical Medicine in London'—it is virtually the history of the origin of Tropical Diseases as a new, special branch of Medicine. The credit for this belongs to Sir Patrick Manson, and it is appropriate that his son-in-law should be the author of this book. He is specially qualified to write it, not merely from filial regard, but because of his personal knowledge of many of the leading characters, and because he himself reached eminence in the subject.

Dr. Patrick Manson worked in China from 1875 to 1889, and his experience led to many new discoveries including the vector transmission of tropical diseases. On his return, he settled in London, where he continued his research and taught the many new principles not then included in the medical curriculum. His fame spread widely, so that notable people from many countries used to visit him. Thus 'Manson's School' started in a small way, in his home. In 1898 he published a manual on 'Tropical Diseases', which furthered the claims of tropical diseases as a separate branch of Medicine.

The necessary hospital accommodation was provided by the Seamen's Hospital Society—first on the so-called 'Dreadnought' ships anchored off Greenwich, and later at the large Greenwich Hospital on shore. In 1890, a branch hospital was erected in the London Dock area, and to the staff of this hospital, Dr. Manson was appointed. At that time, the death rate among Colonial officers serving in the tropics was appalling. So, with the active backing of Mr. Joseph Chamberlain, he not only enlarged this hospital, but succeeded in establishing the London School of Tropical Medicine attached to it. Instruction was given to Colonial officers in 3-monthly courses 3 times a year, and later a diploma was granted.

After the 1914-18 War, because of the isolated position in the dock area, the hospital and school was transferred to central London. No sooner was this completed, when a basic change took place, namely the combination of hygiene with tropical medicine. With the assistance of the Rockefeller Foundation, the sumptuous new buildings of the London School of Hygiene and Tropical Medicine were opened in 1929. This institution continues to meet a world-wide need.

Much research and a vast store of personal knowledge are the ingredients which make this book so delightful to read. The author has done the profession a great service in preserving the history covering the events and the stories of the many personalities connected with the success of the School of Tropical Medicine in London.

H.R.A.

A NEW JOURNAL OF MEDICAL HISTORY

Medical History. A Quarterly Journal devoted to the History and Bibliography of Medicine and the Related Sciences. Vol. I No. 1. January 1957. Editor: W. J. Bishop. Pp. 90. Annual Subscription £2 10s. Single copies 15s. London: Wm. Dawson & Sons Ltd.

Contents: Blue Books, by Sir John Charles. The Health of James the Sixth of Scotland and First of England, by A. L. Goodall. The Historical Aspects of Venous Thrombosis, by S. T. Anning. John Hunter and Veterinary Medicine, by Ernest A. Gray. The Plague in Cambridge, by Raymond Williamson. Texts and Documents; Society Reports; News, Notes and Queries; Book Reviews; Medical Anniversaries of 1957.

Medical historians are rare birds, but the species is on the increase—if current trends in medical literature are to be trusted. No fewer than 6 journals devoted almost exclusively to medical history and bibliography have been launched in the past year or so, and the enthusiasm of the New World for the subject appears now to be spreading to the European countries which comprise the cradle of Western Medicine.

The last of these journals (and the most likely to stay the course) is a new quarterly, *Medical History*, the first number of which appeared on 1 January 1957. Up to now the Royal Society of Medicine has incorporated the minutes of its Section for the History of Medicine in its own *Proceedings*, but for the most part medical historical papers have appeared (when they are published) in the general medical press. As a consequence they are frequently lost to the specialist in this branch. The new journal's most obvious strength and value will lie in filling this gap, in providing a channel for the substantial volume of research into medical history that is carried on in Great Britain. But one hopes that it will serve a wider purpose. There is a place for an authoritative, discriminating English-language journal of medical history which, by the quality and scope of its contents, can reflect the olympian view of the subject to its readers, and become to them what, say, the *Journal of Anatomy* is to the anatomists. *Medical History* promises to fulfil this role, for it is already the official organ of the Norwegian Medical Historical Society (as well as of the leading English and Scottish societies) and the first number indicates that the editor takes the international view of his subject. London of all the world centres is perhaps best suited for his seat, for the Wellcome Historical Medical Library, with its 250,000 volumes of medical and scientific literature from the earliest days of printing to the present day, is the most comprehensive collection of its kind ever assembled. Of this library it is said that 'Sooner or later all paths in medical history lead to it', and much of the research appearing in the pages of *Medical History* will undoubtedly emanate from it.

The first number, bearing a miniature portrait of Thomas Linacre on the cover, runs to 90 pages. The make-up is reminiscent of *The Practitioner*, although the format is of quarto size and the type-face larger. The illustrations (black and white) are good. Specimen copies of this first number of *Medical History* can be obtained free of charge upon application to the publishers at 4 Duke Street, Manchester Square, London, W.1.

E.H.B.

NERVOUS CONTROL OF BLOOD FORMATION

Die Zentralnervose Regulation des Blutbildes. Von Professor Etsuzo Komiya. VIII + 72 Seiten. 49 Abbildungen. DM 12. Stuttgart: Georg Thieme Verlag. 1956.

Inhaltsverzeichnis: Vorwort. Einleitung. I. Die Lokalisation des Zentrums der Blutkörperchenregulation. II. Der Verlauf der Nervenfasern zur Blutkörperchenregulation und ihre Verteilung in den verschiedenen Organen. III. Der Mechanismus der nervösen Regulation der Blutkörperchen. IV. Die nervöse Regulation der Blutkörperchen und deren Funktion. V. Die nervöse Regulation der Blutkörperchen in ihrer Beziehung zu den Baucheingeweiden. VI. Die nervöse Regulation der Blutkörperchen und ihre Bedeutung für die Klinik Schlussbemerkungen. Literatur.

The author, who is a Japanese Professor of Medicine, began his experiments in this, to me, hitherto unknown field in 1934. The aim of these studies was firstly to find the centres in the central nervous system which control the blood corpuscles. Having done this, the arduous task of establishing the nervous pathways which share in this control began. The most interesting chapter deals with the mechanism involved. Furthermore, the author and his co-workers have endeavoured to prove that not only are the numbers of the corpuscles controlled by the nervous system, but also their function, e.g. the motility of neutrophils. Finally the significance of the results of these experiments to the clinician is discussed. This is an interesting booklet on a very unusual subject.

W.E.K.L.

PROGRESS IN CLINICAL MEDICINE

Progress in Clinical Medicine. Third Edition. Edited by Raymond Daley, M.A., M.D. (Cantab.), F.R.C.P. and Henry Miller, M.D. (Durh.), F.R.C.P., D.P.M. Pp. x + 424. 36 Illustrations. 40s. net. London: J. & A. Churchill Ltd. 1956.

Contents: I. Treatment and Prevention of Infections. II. The Care of the Aged. III. Gastrointestinal Disorders. IV. Metabolic Disorders. V. Cardiovascular Diseases. VI. Renal Disorders. VII. Blood Diseases. VIII. Diseases of the Chest. IX. Diseases of the Nervous System. X. Endocrine Disorders: Cortisone and Corticotrophin (ACTH). XI. Radioactive Isotopes in Medicine. XII. The Chronic Rheumatic and Allied Diseases.

If there is one outstanding feature of this publication it is the balance which is struck between the academic and practical components of the various problems under discussion: in each chapter the well-proven experimental work is presented, followed by a review of the clinical aspects, including details of the orthodox treatment, interpreted in the light of the latest knowledge.

An immense amount of information has been crystallized and concentrated in every chapter, and this is nowhere better exemplified than in the section on renal disorders in which, surprisingly, the complexities of electrolyte disturbances have been dissected in an understandable manner. The chapter on geriatrics is a welcome innovation and it contains information which is not generally known.

The wide range of subjects, written by a panel of experts in a readable flowing style of English without a trace of irrelevancy, covers such a wide field that this small volume is practically a text-book in miniature.

Since only the most authenticated knowledge is presented the book will not become obsolescent as rapidly as most 'recent advances'. Furthermore, it is printed in bold type, on good paper, and the finish is a triumph of the bookbinder's art.

Its special approach, as indicated above, makes it an invaluable publication both for the busy specialist and the busy general practitioner.

One can offer no adverse criticism of this work—only praise and strong recommendation.

H.J.

TROPICAL HYGIENE

Jordan's Tropical Hygiene and Sanitation. Third Edition. By W. Wilkie. Pp. viii + 408. 122 Illustrations. 12s. 6d. net. London: Baillière, Tindall and Cox Ltd. 1956.

Contents: I. Air and Ventilation. II. Water. III. Sanitary Building Construction. IV. Refuse Disposal and House Drainage. V. Food and Meat. VI. Disinfection. VII. Communicable Diseases. VIII. Medical Entomology. IX. The Destruction of Rats. X. Health Education and Propaganda. Appendix I Glossary. Appendix II Royal Society of Health Examination Syllabus. Index.

This little handbook, which has now reached the 3rd edition—a sure indication of its appeal and popularity—sets out most successfully to provide a reference work for the health official in tropical or subtropical countries.

Several minor inaccuracies have, however, been permitted to creep into the test (*vide* reference to the use of the carbolic series as a suitable disinfectant for smallpox (p. 256) and the further surprising statement on the same page that formaldehyde tablets are seldom employed today for the purpose of terminal disinfection.

The print is clear and the language direct and authoritative, two important requisites in any manual designed for those groups whose home language may not be English.

A very useful appendix of building terms has been included, and the index appears adequate.

I have no hesitation in recommending this book to health inspectors in training in tropical and subtropical parts of Africa, and even to lay members of the community who are interested in environmental sanitation.

E.D.C.

CORRESPONDENCE : BRIEWERUBRIEK

TELEPHONE CONSULTATIONS

To the Editor: The question of telephone consultations was discussed at the recent meeting of the Federal Council in the light of a letter giving the opinion of the Executive Committee of the South African Medical and Dental Council. This stated that 'in general, consultations by telephone should be discouraged. The Committee can, however, visualize circumstances under which such consultations would be proper and under which the right of a medical practitioner to make a charge therefor could not be disputed'.

I feel that this matter is one of interest to all practitioners and that they should know and understand the attitude of the Executive Committee of the South African Medical and Dental Council in this regard.

A. H. Tonkin
Secretary

Medical Association of South Africa
Medical House
Cape Town
16 May 1957

VAAL RIVER BRANCH

To the Editor: We wish to draw your attention to the fact that a new Branch has been formed in Vereeniging—the Vaal River Branch—and the following are members of the Branch Council:

President, Dr. W. Chapman, *Vice-chairman*, Dr. E. S. Adderley. *Hon. Secretary*, Dr. A. D. Smit, *Assistant Hon. Secretary*, Dr. C. G. Albertyn. *Other members*, Dr. W. A. H. Chapman, Dr. C. van Heyningen, Snr., Dr. H. Bernstein, Mr. J. Hamman and Dr. A. Folb.

Would you be so kind as to publish this in the *South African Medical Journal*.

A. D. Smith
Hon. Secretary

Vaal River Branch
320 United Buildings
Vereeniging
6 May 1957